

**“CLINICAL STUDY OF RISK FACTORS, CLINICAL PRESENTATION AND  
MANAGEMENT OF CELLULITIS LOWER LIMB”**

**A DISSERTATION SUBMITTED TO THE TAMILNADU  
Dr. MGR MEDICAL UNIVERSITY**

**CHENNAI**

*In partial fulfilment of the Regulations*

*for the award of the Degree of*

**M.S. (GENERAL SURGERY)**

**BRANCH - I**



**DEPARTMENT OF GENERAL SURGERY**

**TIRUNELVELI MEDICAL COLLEGE**

**TIRUNELVELI**

**MAY 2018**

**CERTIFICATE BY THE GUIDE**

This is to certify that the dissertation entitled “**CLINICAL STUDY OF RISK FACTORS, CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB**” is a bonafide research work done by **Dr.S.SENTHUR PANDIAN**, Postgraduate M.S. student in Department of General Surgery, Tirunelveli Medical College & Hospital, Tirunelveli, in partial fulfilment of the requirement for the degree of M.S. in GENERAL SURGERY.

Date:

Place: Tirunelveli

**Dr.J.SULAIMAN**

Associate Professor Department of General Surgery,  
Tirunelveli Medical College,  
Tirunelveli

## **CERTIFICATE BY THE HEAD OF THE DEPARTMENT**

This is to certify that the dissertation entitled “**CLINICAL STUDY OF RISK FACTORS, CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB**” is a bonafide research work done by **Dr.S.SENTHUR PANDIAN**, Postgraduate M.S. student in Department of General Surgery, Tirunelveli Medical College & Hospital , Tirunelveli, under the guidance of **Dr.J.SULAIMAN**, Associate Professor, Department of Surgery, Tirunelveli Medical College & Hospital, Tirunelveli, in partial fulfilment of the requirements for the degree of M.S. in GENERAL SURGERY.

Date:

Place: Tirunelveli

**Dr.V.PANDY, M.S.,**  
Professor and HOD of General Surgery,  
Tirunelveli Medical College,  
Tirunelveli

**CERTIFICATE BY THE HEAD OF THE INSTITUTION**

This is to certify that the dissertation entitled “**CLINICAL STUDY OF RISK FACTORS, CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB**” is a bonafide and genuine research work carried out by **Dr.S.SENTHUR PANDIAN** under the guidance of **Dr.J.SULAIMAN**, Associate Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

Date:

**Dr.K.Sithy Athiya Munarvah,MD., (Patho)**

**DEAN**

Tirunelveli Medical College,

Place: Tirunelveli

Tirunelveli

## **COPYRIGHT**

### **DECLARATION BY THE CANDIDATE**

I hereby declare that dissertation entitled “**CLINICAL STUDY OF RISK FACTORS, CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB**” is a bonafide and genuine research work carried out by me under the guidance of **Dr. J.SULAIMAN**, Associate Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

The Tamil Nadu Dr.M.G.R. Medical University, Chennai shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic/research purpose.

Date:

Place: Tirunelveli

**Dr.S.SENTHUR PANDIAN,MBBS.,**  
Postgraduate in General Surgery,  
Tirunelveli Medical College,  
Tirunelveli

## ACKNOWLEDGEMENT

I am obliged to record my immense gratitude to **Dr.Sithy Athiya Munarvah.** Dean, Tirunelveli Medical College Hospital for providing all the facilities to conduct the study.

I express my deep sense of gratitude and indebtedness to my respected teacher and guide **Dr. J.SULAIMAN,** Associate Professor and **Prof.Dr.V.Pandy,M.S.,** HOD, Department of General Surgery, Tirunelveli Medical College, Tirunelveli, whose valuable guidance and constant help have gone a long way in the preparation of this dissertation.

I am also thankful to Assistant Professors **Dr.Irene Aruna Edwin, Dr.S.Nambirajan, and Dr.K.Sathik,** for their help.

I express my thanks to all of the staff members of the Department of General Surgery and all my Postgraduates colleagues and friends for their help during my study and preparation of this dissertation and also for their co-operation.

I always remember my family members for their everlasting blessings and encouragement.

Lastly, I express my thanks to my patients without whom this study would not have been possible.

Date:

Place: Tirunelveli

**Dr. S.SENTHUR PANDIAN,MBBS.,**  
Postgraduate in General Surgery,  
Tirunelveli Medical College,  
Tirunelveli

# *CONTENTS*

SL. NO.	TOPIC	PAGE NO.
1.	INTRODUCTION	1
2.	AIM OF STUDY	3
3.	REVIEW OF LITERATURE	4
4.	MATERIALS AND METHODS	43
5.	RESULTS	53
6.	DISCUSSION	71
7.	CONCLUSION	75
	BIBLIOGRAPHY	
	ANNEXURE I - PROFORMA	
	ANNEXURE II - MASTER CHART	
	ANNEXURE III- ABBREVIATIONS	

## LIST OF TABLES

TABLE. NO.	DESCRIPTION	PAGE NO.
1.	AGE DISTRIBUTION	53
2.	SEX DISTRIBUTION	55
3.	GRADE OF CELLULITIS	56
4.	LIMB INVOLVED	57
5.	CAUSE OF CELLULITIS	58
6.	MICRO-ORGANISMS CULTURED	61
7.	SENSITIVE DRUGS	63
8.	CIRCULATORY CHANGES OBSERVED	65
9.	TREATMENT	66
10.	OUTCOME	68
11.	MANAGEMENT OF THE WOUND	70



online@tymc.ac.in, tirec@tymc.ac.in; www.tymc.ac.in

Tirunelveli Medical College, Tirunelveli - 627011  
State of Tamilnadu, South India

## **CERTIFICATE - II**

This is certify that this dissertation work title **CLINICAL STUDY OF RISK FACTORS, CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB** of the candidate **Dr.S.SENTHUR PANDIAN** with registration Number **221511358** for the award of **M.S.** in the branch of **GENERAL SURGERY.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **1 percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.



URKUND

Document: [CLINICAL STUDY OF RISK FACTORS CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB.pdf](#) (20081002)

Submitted: 2017-09-19 17:11 (+05:00)

Submitted by: [Senthur pandian s \(senthur\\_pandian@yahoo.co.in\)](#)

Receiver: [senthur\\_pandian@yahoo.co.in](#)

Message: CLINICAL STUDY OF RISK FACTORS CLINICAL PRESENTATION AND MANAGEMENT OF CELLULITIS LOWER LIMB [Link to message](#)

1% of this approx. 22 pages long document consists of text present in 2 sources.

Sources: Highlights

Rank	Path/FileName
1	<a href="http://www.arkivis.se.com/gateway/111113/sensations-clag">http://www.arkivis.se.com/gateway/111113/sensations-clag</a>
2	<a href="http://www.arkivis.se.com/gateway/111113/sensations-clag">http://www.arkivis.se.com/gateway/111113/sensations-clag</a>
3	Alternative sources
4	Sources not used

1 INTRODUCTION Cellulitis is a condition which is characterized by inflammation of connective tissue of the skin with severe involvement of dermal and subcutaneous layers. It is principally a bacterial infection, the organism can be either the normal skin flora or an exogenous one. It involves mostly the skin which is more prone for the break, cracks, blisters, ulcers, cuts, (the wounds or hospital related injuries like surgical wounds or the intravenous cannulae). Lower limbs are the most commonly involved sites as the skin over there is much susceptible for the injuries mentioned. As commonly known, Diabetes are the most susceptible population for the lower limb cellulitis primarily because of the fact they have more incidence of foot ulcers (due to the neuropathy and Vascuopathy which arises in the form of sensory loss and poor distal circulation) and also because they are immunocompromised. Poor glycaemic control aids the growth of the organism in the ulcers they develop and eventually results up in the cellulitis. Yet, there is a significant section of population who are non-diabetics, are also more prone for the development of lower limb cellulitis and its complications. Early cellulitis can be managed in out-patient unit with oral antibiotics, analgesics and treating the primary cause. But cellulitis of higher grades, with its complications like fever, myositis, fetid odour, hospital admission, parenteral antibiotics and surgical management.

5 Aims OF THE STUDY in the study group of 230 cases of lower limb cellulitis our aim is to 1) To study the age and sex distribution of the patients with lower limb cellulitis 2) To analyse various causes/risk factors for lower limb cellulitis 3) To study various grades of presentation of lower limb cellulitis 4) To study the spectrum of infectious agents responsible for cellulitis and its sensitivity patterns 5) To study the distribution of grades of the lower limb cellulitis.

URKUND Report - CL.pdf

## INTRODUCTION

Cellulitis is a condition which is characterized by inflammation of connective tissue of the skin with severe involvement of dermal and subcutaneous layers. It is principally a bacterial infection, the organism can be either the normal skin flora or an exogenous one. It involves mostly the skin which is more prone for breaks, cracks, blisters, ulcerations, cuts, bite wounds or hospital related injuries like surgical wounds or the intravenous cannulae. Lower limbs are the most commonly involved sites as the skin over there is much susceptible for the injuries mentioned.

As commonly known, diabetics are the most susceptible population for the lower limb cellulitis primarily because of the fact they have more incidence of foot ulcers (due to the neuropathy and vasculopathy which ensues in the form of sensory loss and poor distal circulation)and also because they are immunocompromised. Poor glycemic control aids the growth of the organism in the ulcers they develop and eventually results up in the cellulitis.

Yet, there is a significant section of population who are non-diabetics, are also more prone for the development of lower limb cellulitis and its complications.

Early cellulitis can be managed in out-patient unit with oral antibiotics, analgesics and treating the primary cause. But cellulitis of higher

grades, with its complications like blisters, myositis, fasciitis needs hospital admission, parenteral antibiotics and surgical management.

## **AIM OF THE STUDY**

In the study group of 100 cases of lower limb cellulitis our aim is to

- ) To study the age and sex distribution of the patients with lower limb cellulitis
- ) To analyse various causes/risk factors for lower limb cellulitis
- ) To study various grades of presentation of lower limb cellulitis
- ) To study the spectrum of infectious agents responsible and their sensitivity pattern
- ) To study the circulatory changes in the lower limb affected with cellulitis
- ) To study the underlying bone involvement, if any
- ) To analyse the various modes of treatment employed
- ) To study the outcome of the treatment in the study group
- ) To discuss the management of the resultant wound after treating the condition.

## **REVIEW OF LITERATURE**

Cellulitis is defined as the non-necrotising cutaneous inflammatory condition also involving the subcutaneous tissue, manifests with erythema, warmth, pain and swelling, the process actually related to the acute infection. In classical considerations, it is the inflammatory process without the formation of abscess or purulent discharge, or involvement of the underlying muscle, fascia or bones. But the recent texts define cellulitis along with its overlapping complications like frank abscess formation, ulcerations, involvement of the underlying fascia and the muscles.<sup>1,6</sup>

Cellulitis is considered a serious infection that use to spread under the skin affecting the soft tissues and the fat underneath the same. It is not contagious and can affect anyone with the damaged or broken skin. Cellulitis tends to occur in the legs, arms and face. The one which involves the face is more common in the infants and the old people. Periorbital variety occurs around the eye and it is severely mutilating one if left untreated. Whereas in the adults, Lower limbs are the most commonly involved sites<sup>2</sup> as the portal of infection i.e., cracks, cuts, breaks, ulcers are more common in the lower limbs, especially in individuals used to bare foot walking.

Cellulitis, ulcers and soft tissue infections of the lower limb are not uncommon in the non-diabetics, and it is to be noted that they are in increasing trend.

## **RELEVANT ANATOMY OF THE LOWER LIMB<sup>5</sup>**

The lower limb according to the anatomic terminology refers to the evolutionised hind limbs in the human beings. It includes pelvic girdle, buttocks, hip, thigh, lower leg and the foot. It is mainly the locomotory organ unlike the upper limb which is destined for various fine works.

### **VASCULAR ANATOMY**

The major arterial supply of the lower limb arises from the continuation of the external iliac artery in the form of Femoral artery, which covers the majority of the arterial zone in the anterior and the medial aspect of the thighs, along with its branches like deep artery of the thigh, medial and lateral circumflex arteries. Obturator artery, a branch of Internal iliac artery also supplies the same territory of the thigh.

The posterior region of thigh and the gluteal regions are supplied by superior gluteal artery, inferior gluteal artery, internal pudendal artery and the perforating arteries of the thigh.

Lowerdown, in the leg the arterial supply is principally taken care by the popliteal vessel, which is a direct continuation of the femoral artery at level of adductor hiatus which is a part of the adductor magnus. Anterior tibial and the Posterior tibial arteries are the branches of the popliteal artery.



Posterior tibial artery gives rise to the common peroneal or the fibular artery and these three vessels contribute the major arterial supply for the lower leg. The anterior tibial artery continues down as the dorsalis pedis artery, the major artery of foot.

Human beings being bipedal, unlike the four legged animals, the blood has to be propelled up against the gravity in the vertical column to the level of heart. In the lower limbs more than 90% of the flow occurs through the deep venous system.

The arteries in the lower leg are accompanied by their pair of venae comitantes which ramifies in the form of sinusoids in the gastrocnemius and soleal group of muscles, and finally all join together to form the popliteal vein and the same continues above as the femoral vein. This system of veins has been classically explained as the deep venous system of the lower limb.

The superficial venous system carry less than 10 - 15 % of the lower limb venous return and this communicates with the deep principally at two sites, one at the groin and the other behind the knee. There are few to many interconnecting veins called perforators in the calf and thigh which can be either direct or indirect.

The superficial venous system consists of

- Dorsal venous arch of the foot
- The great/long saphenous vein

- The small/short saphenous vein
- Posterior arch vein
- Gia-comini vein
- Other few tributaries

Incompetent valves in the superficial system or the sapheno femoral or saphenopopliteal junction is responsible for venous reflux and this will result in the stasis ulcer. These ulcers can act as the entry point for the organisms and thus leading on to the cellulitis.

## **MUSCULAR COMPARTMENTS OF THE LOWER LIMB<sup>5</sup>**

The knowledge about the fascial compartments is important in such a way that it helps in planning the fasciotomy incisions in conditions where there is a need for decompression so as to avoid the compartment syndrome.

The thigh has been divided into three fascial compartments, each comprising of a specific group of muscles and nerve supplying the group.

The medial fascial compartment of thigh comprises of the hip adductors, (adductor longus, adductor brevis, adductor magnus), gracilis. The obturator nerve is the principle nerve supply for this compartment.

The posterior fascial compartment of the thigh comprises of the flexors of the knee and extensors of the hip. (biceps femoris, semitendinosus, semimembranosus). These muscles (hamstrings) are chiefly innervated by

the sciatic nerve, more specifically the tibial nerve. The fibular nerve innervates short head of the biceps femoris.

The anterior fascial compartment of thigh comprises of extensors of the knee and flexors of the hip. It includes sartorius and quadriceps femoris (which comprises of vastus lateralis, vastus intermedius, vastus medialis and the rectus femoris). The muscles in this compartment of the thigh (also the pectineus of the medial compartment) are supplied by the femoral nerve.

The leg has been divided into four fascial compartments, as the posterior compartment itself has been divided into superficial and the deep compartments. The tibialis posterior muscle at times tend to occupy a separate fascial compartment of its own.

Anterior compartment of leg comprises of Tibialis anterior, Extensor hallucis longus, Extensor digitorum longus, Peroneus tertius. The principle nerve supply for this compartment is the Deep peroneal nerve, and the arterial supply is by Anterior tibial vessels.

Lateral compartment of leg comprises of the Fibularis/peroneus longus, and Fibularis/peroneus brevis. The nerve supply for the muscles of this compartment is derived from Superficial peroneal nerve.

Deep posterior compartment of leg comprises of Tibialis posterior, Flexor hallucis longus, Flexor digitorum longus, Popliteus. Nerve supply to

this compartment is derived from the Tibial nerve, the arterial supply is by the Posterior tibial vessels.

The Superficial posterior compartment of leg comprises of Gastrocnemius, Soleus, Plantaris. Nerve supply for this compartment is from the Medial sural cutaneous nerve.

The foot is composed of five compartments.

The interosseous compartment which is bounded by the lateral first metatarsal in the medial aspect, the metatarsal bones and the dorsal interosseous fascia on the dorsal aspect, and the plantar interosseous fascia in the plantar aspect.

The lateral compartment which is bounded by the shaft of the fifth metatarsal bone dorsally, plantar aponeurosis in the lateral aspect, and the intermuscular septum in the medial side.

The central compartment is bounded by the intramuscular septum in the lateral and medial aspects, the interosseous fascia in the dorsal aspect, plantar aponeurosis in the plantar aspect.

The medial compartment is bounded by the inferior surface of first metatarsal in the dorsal aspect, the plantar aponeurosis extension in the medial aspect, and the intramuscular septum in the lateral side.

The calcaneal compartment comprising of the quadratus plantae muscle.

## **AETIOPATHOGENESIS OF CELLULITIS:<sup>3,4,6</sup>**

Risk factors for the formation of the cellulitis in the lower limb are principally any breaks/cuts or ulcers, for which the lower limbs are more prone.

### **Idiopathic:**

In many cases, the exact causes for the local cellulitis remain unknown.

### **Traumatic Wounds :**

Uncared/ unattended wounds in the lower limb can act as a portal of entry of the organism. Bare foot walking in the tropical countries can lead to trivial trauma and most of which will remain unnoticed.

### **Web space infections and intertrigo :**

Breaks in the web space and intertrigo can be reason for cellulitis in many cases. Despite the fact, that dermatophytes do not cause cellulitis, they lead to formation of scales and fissures forming the port of entry for the bacteria.

### **Bites :**

Human/ animal/ insect/ unknown bites in the lower limb are the potential causes for cellulitis as the wounds of this genre are at immense risk of infection.

**Burns :**

Burns/ scald in the limb will destroy the protective skin and negligence of proper wound management will end up in cellulitis.

**Peripheral vascular disease :**

Due to circulatory impairment the limb is prone for ischemic ulcers and this can act as the nidus for the infection.

**Infected venous stasis ulcers :**

Venous eczema itself is a differential diagnosis for the cellulitis, but in cases where the venous eczema leads on to ulcer and which on infection, will result in cellulitis in its own form.

**Edematous limb :**

Edema in lower limb due to renal failure, congestive cardiac failure, hypoproteinemia or lymphedema can lead on to break in the skin, due to the underlying tension and these breaks act as the port of entry for the infectious agents. Obese are more prone for the edema induced cellulitis, because of easy giving away of tissues, due to the thick fat planes.

**Other causes include :**

- Infection of the bone beneath the skin
- Immunocompromised individuals
- Foreign objects in the limb like I.V. cannulas, draining tubes.
- Inflammatory conditions like Lupus, vasculitis, etc.

- Other rare causes of ulcer foot like Hansen's disease, infected malignant ulcers.

Cellulitis is common in the poor socioeconomic class individuals due to the poor hygiene practices and dense sheltering.

The skin offers an effective barrier against bacterial invasion. Bacterial infections occur once the bacteria invades the small breaks or cuts in the skin. The bacteria which causes the disease will get into the lymphatic system, the network of vessels and nodes, leading on to the spread of infection.

In most cases, the causal entry of the cellulitic organisms can be identified, but in some cases the obvious port of entry could not be found and in these cases, entry could be due to the microscopic changes of the skin or specific invasive qualities of certain bacteria. At times cellulitis occur due to the metastatic seeding of the causative organism from the distant focus of infection as that in the immune-compromised individuals. This case is particularly common in that of the infection due to *Streptococcus*, *Pseudomonas* and *Neisseria* sp.

Elderly patients are at increased risk for the more severe disease.<sup>2,6</sup>. Altered host immune response which occurs in the patients with chronic renal disease, immunodeficiency, malignancy, venous eczema, chronic liver disease and peripheral vascular disease is responsible for the higher

incidence of cellulitis in this group of patients. Scavenger action of the antimicrobial peptides, local control of the immune system by means of Interleukin-dependant neutrophil recruitment and cutaneous integrity for the barrier function against the organisms- all these have significant effects on the individual's defense against infection.

Intravenous and subcutaneous skin popping drug use, also affects the host immunity and the infections occurring in this setting are usually polymicrobial. Usually the infection in the immune altered patients is by the non-group A streptococci, whereas in those with normal host defense mechanisms, the organisms usually responsible are group A streptococci and *Staphylococcus aureus*<sup>7</sup>.

H.influenza type B and *Streptococcus pneumonia* have been previously documented as the causes the cellulitis in the paediatric age group, but now the incidence has come down because of the increasing practice of vaccination against these organisms.

*Streptococcus pneumonia* is related with a malignant form of cellulitis that is frequently characterised by the tissue necrosis, suppuration, invasion into the bloodstream<sup>10</sup>. Even Mycobacterial infections at times can present like cellulitis, these organisms present in the form of subacute to chronic type and are characteristically unresponsive to the shortcourse of routine antibiotics. In these spectrum of individuals the diagnosis is routinely made



by the presence of langhan gaint cells in the granuloma and Acid fast mycobacterial organism from the mycobacterial culture and the biopsy specimens.

In patients with cirrhosis it is the gram negative bacteria which causes the bullous cellulitis<sup>11</sup>. Because of the rapid progression of the disease in these individuals early recognition is important as it can prevent the ensuing shock and death. *Vibrio vulnificus* infection tend to occur more in those individuals with chronic liver disease.<sup>15</sup>

Recurrent incidence of Staphylococcal cellulitis can occur in otherwise immunologically normal individuals in case of nasal carriage of the infectious agent like *Staphylococcus aureus* and association of the individual with Job's syndrome<sup>7</sup>.

Various hospital-acquired infections in the surgical wounds and in the cases of soft tissue trauma can lead on to the local cellulitis. Eventhough cellulitis can be complicated by the formation of abscess, it usually arises from the abscessogenic focus.

Specific organisms are related to the bite injuries apart from the usual cellulitic organisms<sup>16</sup>

- Dog bite wounds- *Capnocytophaga canimorsus*
- Human bite wounds- *Eikenella corrodens*

➤ Rat bite- *Streptobacillus moniliformis*

➤ Cat bite- *Pasteurella multocida*

Punctures and lacerations and other wounds acquired in the aquatic/ damp/ brackish environment are harboured by unusual organisms such as *Aeromonas hydrophila*, *Plesiomonas*, *Erysipelothrix rhusiopathies*, *Mycobacterium marinum*.

*Streptococcus pyogenes*, *Vibrio vulnificus*, and *Aeromonas hydrophila* are generally found to be the Common causes of the monomicrobial necrotizing fasciitis. *C. septicum*, *C. histolyticum*, *C. perfringens*, and *C. novyi* are toxic infectious agents whose super addition to the infectious site can lead on to the gas gangrene.<sup>21</sup>

## **SYMPTOMS OF CELLULITIS:<sup>4,6,11</sup>**

### **Reddening of the skin :**

It can evolve as red streaks or areas of redness. It is often difficult to locate that in darker individuals.

### **Swelling :**

It usually commences from the foot (where the cause is most often present). But can also start from the calf region. It is identified by the stretched tight, glossy appearance of skin.

**Pain :**

Severe, excruciating pain usually will be associated. Area of warm, tender, erythematous swelling in tissues around the existing wound will be seen.

**It can be accompanied by :**

1. Fever
2. Discharge of yellow clear fluid/ pus from the involved area
3. Blistering / bullae
4. Dermal necrosis

With the time, the areas of painful erythema expands (phase of dry cellulitis) as infection is not controlled at the earlier stage. And if this stage is left unattended, this may lead on to small blisters and burst (wet cellulitis).

Also, the untreated infection can lead on to lymphangitis, lymphadenitis or bacteremia.<sup>22</sup> Most often it is unilateral, but can be bilateral at some instances. Evidence says that around 7-10% will eventually go for chronic oedema (also called secondary lymphoedema) and it is because of the damage to the previously well functioning lymphatics.

## COMPLICATIONS :

- Fascitis
- Myositis
- Subcutaneous abscesses
- Compartment syndrome
- Septicemia
- renal failure
- Death

**Necrotising fascitis**<sup>3,8</sup> (class IV cellulitis according CREST grading system for cellulitis<sup>6</sup>), is a form of rapidly progressing soft tissue infection, that involves the subcutaneous tissue and principally the fascia. It has very high incidence of mortality if left untreated. Though it most frequently develops after trauma that compromises the integrity of the skin barrier, it can at instance develop in a healthy individual after a minor injury that does not affect skin barrier such as an ankle sprain.

The spread of infection and its speed is directly related to the thickness of the subcutaneous layer and its movement is along the fascial planes. Various terms have been coined to represent the same and it includes, Melaeny's ulcer, acute dermal gangrene, hospital induced gangrene, hemolytic streptococcal gangrene, suppurative fasciitis and the synergistic necrotizing cellulitis.

The trend is there is being an increase in the incidence of the necrotizing fasciitis because of the rise in the immunocompromised patients such as diabetes mellitus, cancer, peripheral vascular insufficiency, alcoholism, transplant recipients, HIV patients and neutropenia<sup>10</sup>.

The disease is more prevalent in the middle aged and the elderly. And it is very rare in the pediatric age group. Despite the pathophysiology of the disease has not been clearly established, it is thought to be occurring due to multibacterial synergy and symbiosis.

Initially, Group-A beta hemolytic streptococcus has been taken as the major cause of this infection. This as a monomicrobial infection is usually associated with some underlying cause such as immunodeficiency, atherosclerotic vascular disease or venous insufficiency with eczema/edema of the limb. Group-A beta hemolytic streptococcus infections usually affect the limbs and around two thirds of the Group-A beta hemolytic streptococcus infection occurs in the lower extremities.

But now over the last two decades it has been proven beyond doubt that it is a polymicrobial event rather than a monomicrobial infection. Aerobic Gram negative organisms along with anaerobic bacteria colonise the most necrotizing soft tissue infections. In patients with trauma, some medical compromise or recent surgery there develops an environment of local tissue hypoxia which favours the florid growth of anaerobic infections.

Facultative aerobic organisms<sup>12</sup> can grow in these sites because the polymorphonuclear neutrophils show deficit in its functions in sites where there is local tissue hypoxia. The growth of these organisms further reduces the oxidation/reduction potential favouring further anaerobic growth and hence accelerating the disease process.

Carbon di oxide and the water are two principle end products of the aerobic metabolism. Nitrogen, hydrogen, hydrogen sulfide and methane are the gases produced from the combination of the aerobic and anerobic bacterial action in the cases of soft tissue infection. These gases, with the exception of carbon di oxide accumulate in the tissue planes because of the decreased water solubility.

In these cases the group A streptococci and the *Staphylococcus aureus* can occur in isolation or synergistically and are the frequently the initiating organisms. Other spectrum of organisms includes

- *Bacteroides* sp.
- *Enterobacteriaceae* sp.
- *Clostridium* sp.
- *E.coli*
- *Peptostreptococcus*
- *Pseudomonas* sp.
- *Klebsiella* sp.

Bacteroides generally do not cause these infections directly and they are noted as the part of the mixed flora in combo with the infection of E.coli. because it plays the chief role in the alteration of the immune mechanism rather than causing the direct infection. It acts by reducing the local interferon production and this inturn has an adverse impact over the phagocytic capacity of the macrophages and the PMNs.

The spread of organisms from the subcutaneous tissues along the superficial and the deep fascial planes is arguably facilitated by the bacterial enzymes and the toxins they produce. This deep infection can end up in vascular occlusion, damage to the superficial nerves resulting in localized anaesthesia, ischemia, and the tissue necrosis.

Surface protein expression and the toxin production by the bacteria are the important events before the onset septicemia. These favour the adherence of the organism to the tissues and this also protects the bacteria against the effects of phagocytosis by the neutrophils.

Pyrogenic exotoxins produced by the streptococci such SPE A,B,C are known to be directly toxic and these toxins along with SSA (streptococcal superantigen) leads to the release of cytokines and that will result in hypotension. The etiologic agent can also be Staphylococcus aureus harboring the cluster of enterotoxins.

Severe myositis that accompanies the septic necrotizing fasciitis may be due to panton-valentine leucocidin positive staphylococcus aureus strain.

In such cases immunostaining can establish the strong adherence of the toxin to necrotic muscle tissues.

Poor prognosis in the cases of necrotizing fasciitis<sup>13</sup> though been linked to some specific streptococcal strains, McHenry et al found that the monomicrobial infection with *Streptococcus pyogenes* was not having direct association with the increased mortality.

One more study from Hsiao et al states that *Aeromonas* infection, vibrio species infection, presence of malignancy, hypotension, and occurrence of band form WBC count more than 10% are found to be independent predictors of the mortality whereas streptococcal and staphylococcal infections were not found to be the predictors of mortality.

Pre existing liver cell failure, chronic kidney disease, thrombocytopenia, decreased albumin levels and the need for the post operative ventilatory support all represent poor outcome factors in cases of monomicrobial necrotizing fasciitis<sup>14,15</sup>.

Diagnosing the case of necrotizing fasciitis can be difficult and it requires a high level of suspicion. In most cases antecedent trauma or a history of surgery over the region can be noticed.

Olafsson et al put forth that the hallmark sign of the necrotizing fasciitis is the tenderness over the involved skin and the muscle underneath. The intensity of the pain is so much that one would suspect a torn or ruptured muscle. Pain may be out of proportion to the physical findings.



Over the next few hours to days, the pain progresses to anaesthesia due to the damage to the superficial nerve fibres. Other few findings include edema extending beyond the zone of erythema, vesicles in the skin and crepitus.

**Compartment syndrome**<sup>20,23</sup> is a limb and life threatening condition, which is defined as the compression of blood vessels, nerves, and muscle inside the closed compartment within the body. This can lead on to cell death due to the lack of oxygenation because of the blood vessels being compressed by the increased pressure within the muscular compartment. It most commonly involves the forearm and lower leg. Also this can be classified into acute, subacute, and chronic compartment syndrome. The alternative definition for the compartment syndrome, according to Rankin, it is characterized by increased pressure within the closed space and hence compromising the circulation and the function of the tissues in that compartment.

The cause of compartment syndrome in cellulitis is due to the circumferential cellulitis which leads on to excessive pressure on the muscle compartments. The swelling of the tissue within the fascial planes forces pressure upon the muscular compartments, which has only a limited volume. Because of this raised pressure, the small venules and the lymphatic vessels that use to drain the muscular compartments are compressed, and are hence prevented from draining. As the arterial inflow

persists while the outflow is lowered, the pressure shoots up within the muscular compartments. This pressure will finally reduce the amount of blood flow to the capillary bed, and thus causing the tissue to become hypoxic and finally ischaemic. The tissues will release inflammatory factors and this leads to the formation of edema. Untreated and unnoticed compartment syndrome-induced ischemia of the muscles and nerves will eventually lead on to the irreversible damage and the death of the tissues within the fascial compartment. Compartment syndrome is most often a clinical diagnosis. But, it can be tested by gauging the pressure limit within the fascial compartments. If the pressure is sufficiently high, a surgical intervention in the form of fasciotomy is required to relieve the pressure. Various literature suggests that the intracompartmental pressure  $>30$  mmHg as an indication for fasciotomy while others advise a  $<30$  mmHg difference between compartmental pressure and diastolic blood pressure. This second measure is somewhat more sensible in the light of the recent advances in the permissive hypotension, which can allow the patients to be kept under a hypotensive state in resuscitation. It is now somewhat easier to measure the compartmental and the subcutaneous pressures using some sort of pressure transducer modules which are connected to the most modern anaesthetic machines. Most often compartment syndrome is diagnosed through a diagnosis of its inciting cause and not the syndrome itself.

## **GRADING OF THE CELLULITIS :<sup>6</sup>**

**(Clinical Resource Efficiency Support Team-CREST:2005 )**

### **Class I**

Patient will not have signs of systemic toxicity or any comorbidities and are routinely treated with oral antibiotics in the outpatient basis.

### **Class II**

Patient

- a) Have systemic illness
- b) Do not have any systemic illness but have some comorbidity like
  - i. Peripheral vascular disease
  - ii. Chronic venous insufficiency
  - iii. Morbid obesity

Which can affect the resolution of infection.

### **Class III**

Patient have

- i. Significant systemic problems
- ii. Unstable comorbidities
- iii. Limb threatening infection due to  
vascular compromise

#### **Class IV**

- i. Severe life threatening infections like necrotising fasciitis.
- ii. One which is associated with Sepsis syndrome.
- iii. Cellulitis in Immunocompromised individuals.

#### **MANAGEMENT OF LOWER LIMB CELLULITIS<sup>6,16,17,20,23</sup>**

Cellulitis is often a clinical diagnosis and there is no specific investigation which can point towards it as the principal diagnosis.

##### **Laboratory investigations :**

In general no work up is essential in the class I patients and in other few uncomplicated cases of cellulitis in case if the following criteria are met.

- Area of involvement is minimal
- Minimal Pain
- No risk factors to suspect serious illness (generalized disability, extremes of age, immunocompromised individuals)
- No systemic signs of illness (dehydration, dyspnea, chills and fever, altered mental status, fall in the blood pressure)

In all the other patients, diagnostic work up is essential for a comprehensive management of the condition. But no work up should delay the treatment in these individuals.

## **Culture from any skin break / ulceration / blister fluid and its antibiotic sensitivity**

This is of utmost importance because of the prevalence of resistant strains is on the increasing phase and hence mere empirical antibiotic should as far as possible be avoided in the management of the cellulitis.

But in these cases the culture in the form of swab or aspiration or in the form of punch biopsy of the inflamed area, the yield is less than 60%. Deeper tissue samples, collected during the time of the surgical debridement, are required to obtain proper cultures for microorganisms. At the time of incision and drainage being performed, the aspirate or pus collected will have the higher yield of about more than 90%

*Staphylococcus aureus* and Group A streptococcus are the most common organisms cultured in the cellulitic population worldwide, because of the presence of these common organisms in the skin surface and once there is some insult to the skin surface, there is every chance of these organisms to get into the skin to result in the cellulitis.

If the recurrence of cellulitic episodes are suspected to be secondary to fungal infections like tinea pedis or onychomycosis, mycologic evaluation is advised.

Tissue biopsy is not routinely employed but can be performed in the attempt to rule out the non-infectious entity. The tissue section staining and microscopy reveal the findings of inflammation in the soft tissue. Dilatation

of the capillaries, leukocyte infiltration, and bacterial tissue invasion are observed.

### **Blood investigations :**

#### ➤ Complete blood count

Usually tends to show leukocytosis in the setting of the severe disease, but it is not uncommon to see leucopenia in cases of severe disease, especially where the cellulitis is toxin mediated.

Erythrocyte sedimentation rate are frequently elevated especially in individuals with severe disease and those who need prolonged hospitalization.

#### ➤ Renal parameters

Blood urea

Serum creatinine

These two are essentially required to assess the baseline renal function so as to correctly direct the antimicrobial agents.

### **Doppler study of the arterial and venous system**

The arterial and venous system should be evaluated for purpose of assessing distal circulatory changes in terms of arterial flow, venous stasis, venous reflux and most importantly to rule out the Deep vein thrombosis as it can masquerade the symptoms of cellulitis.

These circulatory changes apart from altering local oxidation/reduction potentials also has an impact on the immune processes in the local milieu.

### **Plain X-ray of the affected limb**

This is principally done to rule out the osteomyelitis in the local site which resemble the symptoms of cellulitis or the same is responsible for the cellulitis in the limb. Also this helps to study the site of entry of organisms, which may be harbor the destroyed part of bone due to a trauma or a gangrenous process due to the circulatory impairment.

### **Other radiological investigations**

Current data suggests that ultrasound may have a role in locating an occult abscess and in planning the direction of care, especially in the emergency setting. USG-guided aspiration of the pus has been proven to shorten the hospital stay.

In cases in which the necrotizing fasciitis is the concern, computer tomographic imaging is employed to rule out the condition in patients who are stable. Magnetic resonance imaging can be a part of the work up but MRI typically takes much longer time than that of the CT scanning.

Cellulitis is usually managed conservatively with antibiotics; surgical indication in cellulitis comes to play only when there is circumferential cellulitis (compartment syndrome can be the sequelae), evolving bullae, bronzing of the skin, setting of crepitus and the one which evolves rapidly,

symptoms (pain) discordant with the examination findings. Cellulitis with underneath abscess formation requires surgical drainage.<sup>6</sup>

### **Class I**

These patients can be managed under the primary care set-up by conservative line of management on out patient basis which includes;

- Broad spectrum oral antibiotics
- Limb elevation to reduce the oedema
- Liquid paraffin for topical application once the oedema subsides and skin becomes scaly
- Analgesics can be prescribed for the pain

In the management of the these patients in the outpatient basis the antibiotic regimens are more than 90% effective. The frequent oral antibiotic considerations include

- Dicloxacillin
- Cephalexin
- Amoxicillin

In cases of patient with penicillin allergy employment of the macrolide antibiotic should be considered. Azithromycin and clindamycin are the preferred choices from this group of drugs.



Usage of fluroquinolones are better reserved for the culture proven Gram –ve infections because of the threat of emerging resistance.

Recurrent cellulitis documented in the outpatient department could be due to the circulatory impairment, venous or lymphatic obstruction, immunodeficiency and are most commonly due to the streptococcal infections. Hence for those cases the recurrence should be addressed preferably with the drugs like erythromycin or amoxicillin.

In cases where Tinea pedis is found to be the inciting cause, one should not hesitate to employ the topical or systemic antifungals to combat the same.

In cases of bites, mostly the infection tends to be polymicrobial and so the empirical treatment with antimicrobial agent against the anerobic organisms in addition to the comtemporary antibiotics should be considered.

In such cases, the antibiotics for consideration includes,

- Trimethoprim- sulfmethaxazole
- Amoxicillin- clavulinic acid
- Fluroquinolones

This should be combined along with Metronidazole.

Candidates with lacerations or breaks encountered in the aquatic environment should get antibiotics specific for the organisms which colonise in the brackish water apart from the routine agents. Ideal choices for these individuals include

➤ Doxycycline

➤ Ceftazidime

➤ Cephalexin

Those who have acquired the infection from the fresh water should be considered for 3<sup>rd</sup> or 4<sup>th</sup> generation cephalosporin like cefipime and ceftazidime along with a fluoroquinolone.

*For patients treated on the outpatient basis, the following facts must be borne in mind during follow-up*

) Erythema may increase transiently over the first day of treatment and it simply represents the inflammatory reaction to the cell lysis caused due to the antibiotics

) Patients should be reassessed with the short interval of 2 or 3 days to ensure the improvement

If there is appreciable improvement the antibiotic regimen has to be continued till the inflammatory symptoms subside.

If there is no improvement, one has to consider antibiotic resistance in those individuals and a change in antibiotic should be employed

) If there is progression of the disease or there is development of the systemic symptoms, hospital admission has to be considered in those individuals.

## **Class II**

- Appropriate analgesia for the pain relief
- Antipyretics for any pyrexia
- Hydration has to be ensured (either intravenous or oral )
- Culture directed parenteral antibiotics is mandatory for all patients with class II to class IV cellulitis.
- Elevation of the limb.

The recommendations for the IV regimens for the hospitalized patients include clindamycin and penicillin therapy especially for the severe group A streptococcal and in the cases of clostridial necrotizing infections. In cases, where there is mixed necrotizing infections, with the presence of gas in the deeper tissues, the selection of antibiotic should be in such a way to direct against the aerobic gram positive or the gram negative and anaerobic organisms.

Empirical antibiotic recommendations as per Infectious diseases Society (ISDA) include

- IV Ampicillin-sulbactam 1.5-3.0g 6-8 hrly (in patients with history of penicillin hypersensitivity, metronidazole or clindamycin along with an aminoglycoside or fluoroquinolone)
- IV piperacillin tazobactam 4.5g 6-8 hrly along with IV ciprofloxacin 400mg 12hrly.

- IV Imipenam-cilastatin 1g 6-8 hrly
- IV meropenem 1g 8hrly
- IV cefotaxime 2g 6hrly with metronidazole 500 mg 6hrly

Management principally lies on the care of the local site. In case if there is blistering and there is a potential for the same to burst spontaneously proactive management is suggested which consists of aseptic aspiration with or without deroofing of the blister.

Tension and swelling in some cases lead on to ulceration and there is loss of large amount of exudate. In some class II and all class III and class IV patients, there is loss of protective skin barrier and so the patient is more prone for dehydration. Dressing either with foam or alginate or hydrofibre is employed so as to avoid the maceration of the skin.

### **CLASS III**

- Correction of the co-morbid and management of the local pathology, both should go hand in hand
- Sloughed out and macerated skin should be thoroughly debrided, and underlying abscess if any should be duly drained.
- The principle management lies in the appropriate use of antibiotics and correction of the metabolic status. Culture directed parenteral antibiotics must be employed and culture should be repeated atleast once in two weeks.

Andreason et al advises full thickness excision of tissue, even in normal appearing skin zone, because they have found that these tissues had extensive early vascular thrombosis and vasculitis.

#### **CLASS IV**

Necrotising fascitis as such is considered as a surgical emergency, and it involves close monitoring of the hemodynamic parameters and it mandates aggressive resuscitation. The patient should only be managed in the center, where there is skilled surgical staff for doing extensive debridement / reconstructive surgery.

In a study, Rouse et al the overall mortality rate was 73% i.e., 20 out of 27 patients. Study says prompt recognition and treatment of the condition is essential. Of patients whose treatment was delayed for more than 12 hours, 11 patients expired.

In another study by McHenry et al, the average time from the admission to operation was around 90 hours in the nonsurvivors of necrotizing infection of the soft tissues, and in the cases of survivors, this average time was 25 hours. This indicates that the early intervention and debridement of the infection was associated with a significant reduction in the mortality.

The surgical debridement should be continued till the necrosis ceases and the observation of fresh viable tissue. In cases of irreversible necrosis / gangrene / life threatening sepsis, amputation may be necessary.

(Eventhough, the literature advises, hyperbaric oxygen therapy, it should never defer / delay the surgical management, which ensures higher likelihood of survival).

Extensive debridement of all the necrotic devitalized tissues, rapidly improves the clinical outcome.

Surgical regimen should include

- Surgical incisions should involve and also extend beyond zone of necrosis until the fresh viable tissue is reached
- Wound should be well irrigated
- Perfect hemostasis should be attained
- Debridement and evaluation of the wound should be repeated on the daily basis and must only be done in the operating room

## **THE TECHNIQUE OF FASCIOTOMY<sup>18,19</sup>**

### **Fasciotomy technique for the lower leg**

The Soleus muscle takes its origin from the tibia and the fibula for the entire length of proximal half of the leg. Hence, underneath this “soleus bridge” is the deep posterior compartment of the leg and so its contents are not in the subcutaneous plane.

And so, for the fasciotomy of deep posterior compartment of the leg, the soleus origin should be detached from either of the two long bones.

Proximal to this area, the pressure can be measured through the origin of soleus from the tibia.

Essentially two techniques can be followed when doing a four compartment fasciotomy.

1. Single lateral incision or the Perifibular approach
2. Two incision technique

Though the subcutaneous fasciotomy is appropriate in cases of exertional compartment syndromes, it is not useful for decompression of the deep posterior compartment as this compartment is subcutaneous only in the distal half of the leg.

The length of the incision made has an direct impact on fascial decompression in the leg in cases associated with acute compartment syndrome.

Limited incisions favoured by few authors, claim low morbidity, while others recommend quite longer incisions, emphasizing the fact that they are required to decompress involved compartments efficiently.

Cohen et al described the impact of the length of the incision used in posttraumatic compartment syndromes of the lower limbs managed with fascial decompression using this two-incision technique.

Longer incisions add too little impact of morbidity and influence neither the late functional result nor complication rate. Longer incisions also decrease the risk of the skin acting as the unrecognized compartmental envelope, which is especially needed during the hyperaemic period that follows decompression of an ischemic compartment.

## **Single incision technique**

Whitesides is the one who described this perifibular single incision technique.

A straight lateral incision to the posterior and parallel to the fibular bone right from the level of the fibular head to the point just above the tip of lateral malleolus.

Near the proximal end of the incision, it is important to expose and protect the common peroneal nerve.

The dissection has to be deepened so as to incise the fascia between the soleus and that of the the flexor hallucis longus distally. Then it is extended proximally to release the soleus origin from fibular bone.

This method allows access to the full length of both superficial posterior and deep posterior compartments of the leg.

The incision must be made longer, because the decompression of the deep posterior compartment of the leg is more difficult with this type of dissection than the one from the medial side.

The anterior leading edge of the incision is then retracted so as to expose the anterior and lateral compartments, with exercising meticulous care to negotiate the superficial peroneal nerve as it exits out of the fascia of the lateral compartment and courses anterior in the distal third of the leg.



## **Two incision technique**

This technique adequately decompresses all the four compartments through two incisions,

- Anterolateral incision
- Medial Incision

### **Anterolateral incision**

Longitudinal incision made to reveal the contents of the anterior and the lateral compartment of the leg, taking care to preserve the superficial peroneal nerve in the distal aspect.

### **Medial Incision**

The site of the medial skin incision is important. The maximal bulk of the musculature in the superficial posterior compartment of the leg is proximal and it needs a proximal extension of the incision so as to adequately decompress the compartment.

But, maximal bulk of the deep posterior musculature is seen in the distal half of the limb. The medial incision should be done in a longitudinal manner slightly to the posterior of tibia.

The superficial posterior compartment should then be opened, and the soleus should be detached from its tibial origin so as to expose the deep posterior muscular compartment in the proximal half of the leg.

### **Fasciotomy technique for the thighs:**

- Lateral incision should be made from the greater trochanter to the lateral condyle of the femur
- Iliotibial band should be incised and the vastus lateralis muscle is reflected off the intermuscular septum bluntly, thus releasing the anterior compartment
- Intermuscular septum should then be incised for the length of the incision, so that releasing the posterior compartment. care should be exercised that this release of intermuscular septum must not be made too close to the femur because there are series of complex perforating arteries passing through the septum from posterior to anterior close to the bone.
- Then the medial adductor compartment should be released through a separate anteromedial incision

### **Fasciotomy technique for the foot**

#### **Dual dorsal incisions**

- One incision is placed slightly medial to the second metatarsal bone, thereby reaching between the first and second metatarsals into the medial compartment and also between second and third metatarsal bones into the central compartment of the foot.

- Second dorsal incision should be made just lateral to the fourth metatarsal bone, reaching between the fourth and the fifth metatarsal bones into the lateral compartment of the foot.
- So as to spare the dorsal soft tissue of foot, single incision medial fasciotomy can be used . This medial approach to the foot is made through the medial compartment, thereby reaching across the central compartment into the interosseous compartment dorsally and the lateral compartment and thus releasing all the way across the foot

### **Management of Fasciotomy Wound<sup>19</sup>**

Management of the fasciotomy wound still remains controversial. In a study Andrew et al, Wound complications were documented in around 51% of the patients who had either the primary or delayed primary closure when compared with 5% who had split thickness skin grafts. If all the devitalised tissues have been convincingly excised we can favour immediate coverage with meshed, split thickness skin grafts obtained with a foam vacuum suction dressing. The cosmetic appearance can be improved by subsequent revision of the scar.

- After the fasciotomy is performed, wound should be debrided of all sloughed out and devitalized tissue.
- Must be Treated as the war wounds i.e., without early closing and the coverage should be done with sterile dressing.

- Closure of the fasciotomy wounds can be delayed until the exit from arduous environment.
- Options for the Closure of the wound
  - Delayed primary closure of the wound
  - Healing by the secondary intention
  - Split skin grafting or a suitable flap coverage
- Time for Post operative relook should be individualised according to the wound. closure 3 to 5 days.
- When there is impending muscle necrosis, relook should preferably be done at 24-48 hrs.
- When the edges of the wound do not oppose easily during the attempt of delayed primary closure, split-thickness skin grafting should be considered.

### **Limitations of Fasciotomy**

Fasciotomy is not a benign procedure, and the evidence implies that they can lead on to chronic venous insufficiency due to the impairment of the soleal muscle pump. Also the role of fasciotomy in the cases of compartment syndrome which have been noticed at a late stage i.e., after 8 hours is questionable. It is because of the fact that established myoneural deficits less often recover after the fasciotomy.

Furthermore, fasciotomies which are performed after 35 hours from the time of injury are invariably associated with some sort of severe infection and eventually can even result in death.

## **MATERIALS AND METHODS**

This study, principally an prospective case series study, includes 100 patients who got admitted for lower limb cellulitis and its complications, under all surgical units of Tirunelveli Medical College Hospital, as the study group.

### **PERIOD OF STUDY:**

The study period involves a span of one year and eight months.

### **MATERIALS:**

#### 1) Clinical evaluation

---

- Age incidence
- Sex incidence
- Limb involved
- Severity during presentation

#### 2) Investigations

---

- Complete blood count
- Swab/ wound biopsy culture from the wound
- Study of sensitivity pattern for the organisms
- Doppler study of the arterial and venous system of the involved limb
- Plain X-ray of the limb

3) Management of cellulitis according to its severity

4) Management of the sequelae

## **METHODOLOGY**

All the 100 patients have been studied systematically which started with careful analytical history taking.

- Categorizing the patients according to the age group, and in our study we have included individuals of age group 13-90 years as our study group, among these the incidence of the disease in each of six decades have been recorded.
- Categorizing the patients according to the Gender, to study the incidence of the disease in each sex.
- History regarding the presenting illness, pain, reddening of the region, swelling of the local part, any ulcerations, blister/ bleb formation.
- Whether he or she is aware of the cause for the cellulitis,
- If he or she is not aware of the cause, eliciting history regarding any trivial trauma, unknown bites, history of bare foot walking is considered
- Whether there is history of any such illness previously and if yes how it was managed then, principally to study the incidence of recurrent cellulitis
- Whether the patient is a smoker or an user of tobacco in other forms.

- Whether the patient is having any comorbid medical illness, (hypertension, epilepsy, cardiac illness, chronic kidney disease, chronic liver disease, bronchial asthma)

In the clinical examination, general examination of the patient for the presence of the anemia and jaundice has been studied and the nutritional and hydration status of the patient has been recorded.

Thorough examination of the cardiac system, respiratory system done and the findings recorded. Abdominal examination and central nervous system have also examined. Vital parameters- pulse rate, blood pressure, respiratory rate and temperature have been recorded for all the patients.

Regarding the examination of the lower limb, the limb is studied regarding the

- Extent of cellulitis
- Blisters/blebs
- Presence of subcutaneous abscesses
- Presence of ulcerations
  - To be studied regarding etiology
  - Presence of serous or purulent discharge
  - Status of healing
  - Depth of involvement



- Careful examination for any web space infections, cracks, breaks or cuts
- Involvement of the deeper tissue like muscles, fascia or bones as assessed by the inspection and palpation.
- Distal pulsations and capillary refill time.
- Careful assessment for the threat of compartment syndrome in cases of extensive or circumferential cellulitis, with its classical parameters
  - Disproportionate pain to the presenting clinical symptoms
  - Altered sensations
  - Onset of pallor due to decreased distal flow
  - Weakness of the limb
  - Decreasing pulse volume
  - Symptom of pressure
- The severity involvement of the limb is graded as per the CREST guidelines for grading of cellulitis, which is studied as follows,

**Class I** : Patient will not have signs of systemic toxicity or any comorbidities and are routinely treated with oral antibiotics in the medical or surgical outpatient departments.

**Class II** : Patients either have systemic illness, in the form of fever due to the infective focus or do not have any systemic illness but have some comorbidity like Peripheral vascular disease, Chronic venous insufficiency, morbid obesity which can affect the resolution of infection.

**Class III** : Patients have either significant systemic problems or limb threatening infection due to the vascular compromise, presenting with edema and devitalizing changes in the site or the patient with unstable comorbidities.

**Class IV** : patients in this grade includes those with severe life threatening infections like necrotising fasciitis or with the cellulitis which is associated with Sepsis syndrome. Cellulitis occurring in the Immunocompromised individuals also comes under this category.

After stratifying the patient according to the history and the clinical examination, with relevance to the age, sex and the grade of cellulitis, patients have been investigated as follows,

## **LABORATORY INVESTIGATIONS:**

### **Complete blood count:**

This helps in offering data about the haemoglobin status of the individuals, which has the impact in the rate of improvement of the patient and the Differential count, which often tend to show a shift to the left, in cases of infections and where an active inflammatory response is going on to combat the same.

**Renal parameters:**

Especially the blood Urea and serum creatinine are studied primarily to record the baseline renal function of the patient at the time of admission. Some patients in the study who are the known case of chronic kidney disease, maintenance of the renal parameters during the course of management had the major impact on the outcome of the patients, whereas in the others, it helps to monitor the hydration status of the patient and most importantly to alarm about altered renal function or the possibility of acute renal shutdown during the management of the cellulitis.

Other blood investigations include liver function tests including the coagulation profile, serum protein, serum electrolytes, and HIV and HbSAg assays

**Culture and sensitivity:**

In those individuals who have any obvious blisters or blebs or any discharging fluid from the surface of the cellulitic portion of the involved, a swab is taken from the site for culture and sensitivity pattern is studied.

As the swab culture has poor yields especially where then the swabbing being done from the superficial blisters, and hence we have tissue biopsy culture to study the organism involved.

And in cases where emergency wound debridement and abscess drainage or fasciotomy is needed, the spectrum of organisms and its

sensitivity pattern is studied from the pus / fluid collected during the procedure.

### **Doppler study of arterial and venous system:**

Doppler study is principally to know about the circulatory status in the lower limb which has the major role in the management of cellulitis. With this we studied about the arterial changes in the lower limb, vessels with alterations in the flow pattern, level of arterial involvement and also in cases where an arterial ulcer in the foot or the toe is the entry point for the infectious agent responsible for the cellulitis.

Venous Doppler helps in identifying the venous reflux and venous stasis ulcer, which can also be the portal of entry for the organisms. Its major importance lies in the fact that it rules out the Deep venous thrombosis, which can simulate the cellulitis creating confusion at the diagnosis.

### **Plain X-ray of the affected limb**

This has been done for two reasons, one, to study about any underlying bone involvement in severe forms of cellulitis, second it helps to rule out the cases of osteomyelitis which presents in the form of cellulitis. This also helps to study the infectious entry site as in the cases of arterial ulcers or the trauma whether there is any local bony destruction.

## **TREATMENT**

All patients who presented in the early stage of cellulitis have been started on parenteral antibiotics, covering gram positive, gram negative and anaerobic organisms. The antibiotic response is monitored and if the disease improves, the patient is maintained on the same antibiotic for seven days. If the patient shows no response or if there was progress in the disease, change in the antibiotic was practiced awaiting the culture and sensitivity pattern.

### **SURGICAL INTERVENTION:**

In case of blistering or bleb formation, there is a potential for spontaneous rupture and in those cases proactive aseptic aspiration with or without deroofing of the blister was done. Tension and swelling at the site of cellulitis can lead on to ulceration and there is loss of large amount of exudate. In most patients, there is loss of protective skin barrier and so the patient is more prone for dehydration .steps were taken to correct the hydration status and wound dressing is done so as to avoid the maceration of the skin.

In cases of advanced cellulitis, which is evidenced by devitalized bronzy skin, with underlying pus-pointing, with the suspicion of a subcutaneous abscess, sloughed out and macerated skin are thoroughly debrided, and underlying abscess if any is drained.

Surgical regimen practiced is such that, the surgical incisions involved and also extended beyond the area of devitalized skin until the zone of fresh viable tissue is reached, abscesses if any are drained, fasciotomy was done when there was a threat of compartment syndrome. The wounds were well irrigated and hemostasis attained. Any blood loss encountered during the surgical procedures, was dealt with adequate blood transfusion.

Evaluation of the wound has been repeated on the daily basis and if needed, further debridement has been done.

Those patients with arterial ulcers, cellulitis due to snake bites and few patients with trauma showed gangrenous changes at the site and showed bony destruction and hence in those individuals amputation as necessary was done so as to remove the persisting port of infection.

Patients with life-threatening necrotizing cellulitis, with the threat of impending sepsis syndrome, and with distal decreased blood flow were taken for amputation at appropriate levels.

Of all the 100 patients managed, the outcome of the management has been analysed and recorded. The outcome has been recorded in such a way whether the patient had an uneventful recovery, or the patient had developed tissue loss with the resulting raw area which needed further management, or the patient had a residual deformity or the patient had died because of the disease.

Management of the resultant surgical wound has also been studied. It was done with either delayed primary closure or we allowed the wound to heal by the secondary intention. In cases, where the wound edges are unlikely to get approximated for the healing, and those which resulted in the raw area, split thickness skin grafting has been done.

#### **LIMITATIONS OF THE STUDY:**

- As almost all the patients with early forms of cellulitis (grade I) are managed with oral antibiotics and analgesics in the outpatient basis and this study principally being conducted in the hospitalized patients, this study do not cover the patient with milder grades of cellulitis.
- We do not have facilities for anaerobic culture in our microbiology laboratory, and hence the incidence of anerobic infections could not be studied.

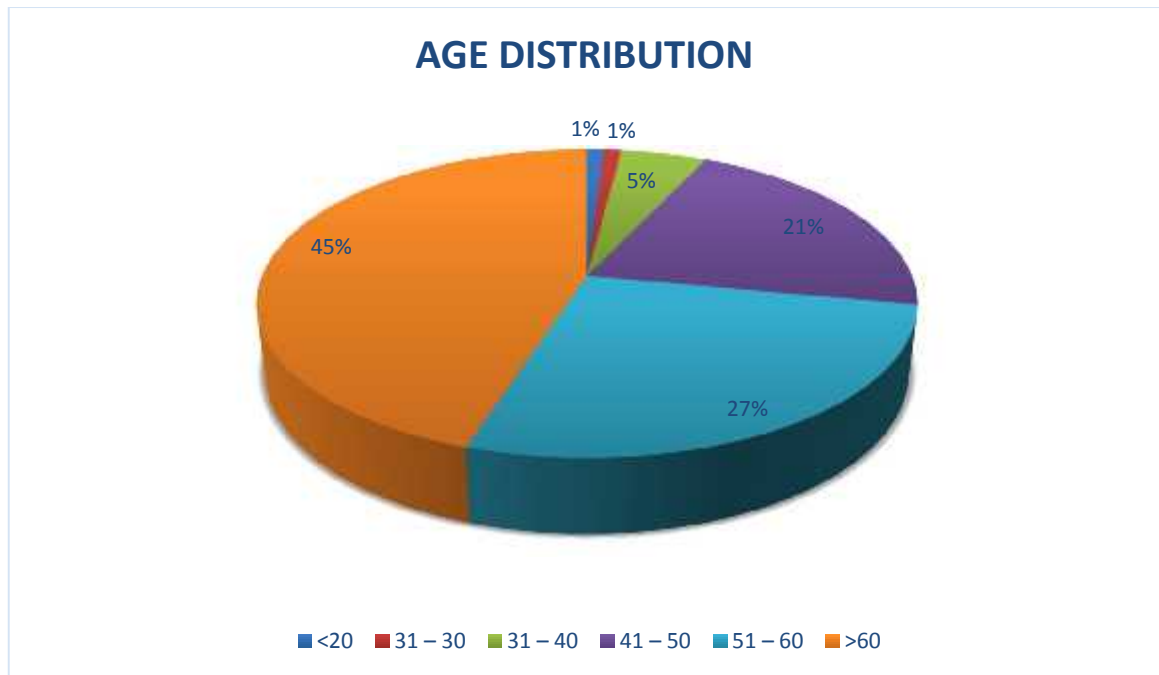
## **RESULTS & OBSERVATIONS**

The following findings were observed in our analytical study regarding the management of lower limb cellulitis, conducted in Tirunelveli Medical College. Totally 100 such patients were included in the study and the result being analysed as follows.

### **1. AGE DISTRIBUTION**

<b>Sl.No.</b>	<b>Age Group</b>	<b>No of cases</b>
1.	<20	1
2.	31 – 30	1
3.	31 – 40	5
4.	41 – 50	21
5.	51 – 60	27
6.	>60	45

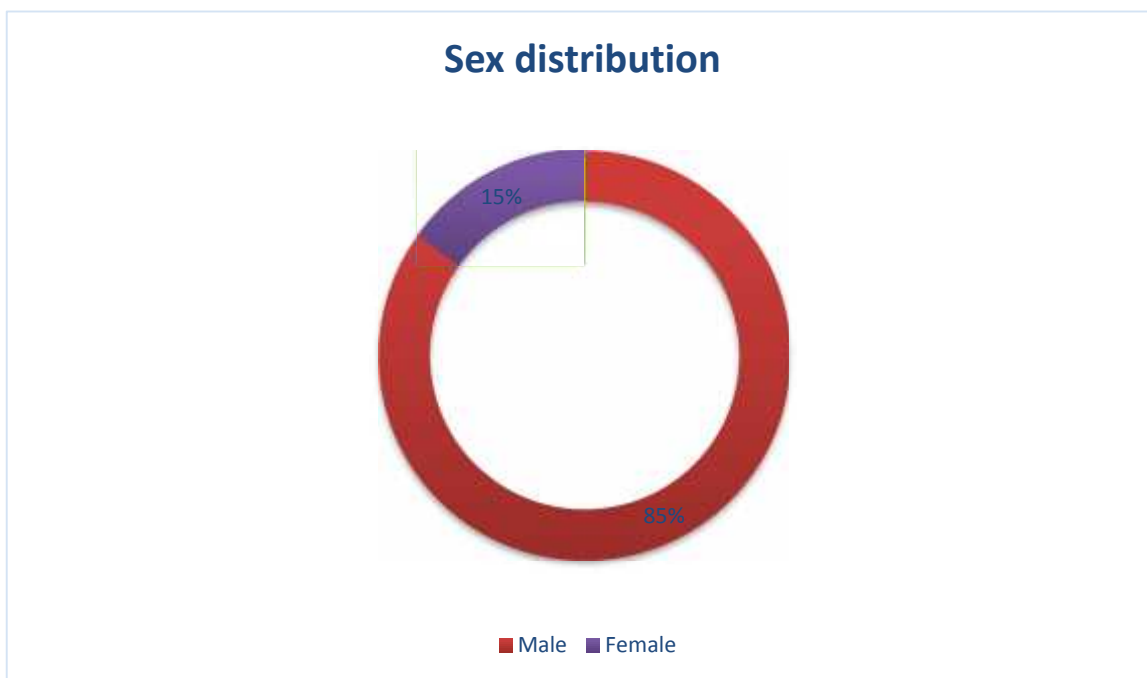




Out of the 100 patients studied 1 belonged to <20 years and 21-30 years, 5 belonged to the age group 31-40years, 21 belonged to 41-50 years, 27 belonged to 51-60 years and 45 were from the age group more than 60 years from which it is evident that as the age increases, the incidence of cellulitis increases.

## 2. SEX DISTRIBUTION

Male	Female
85	15



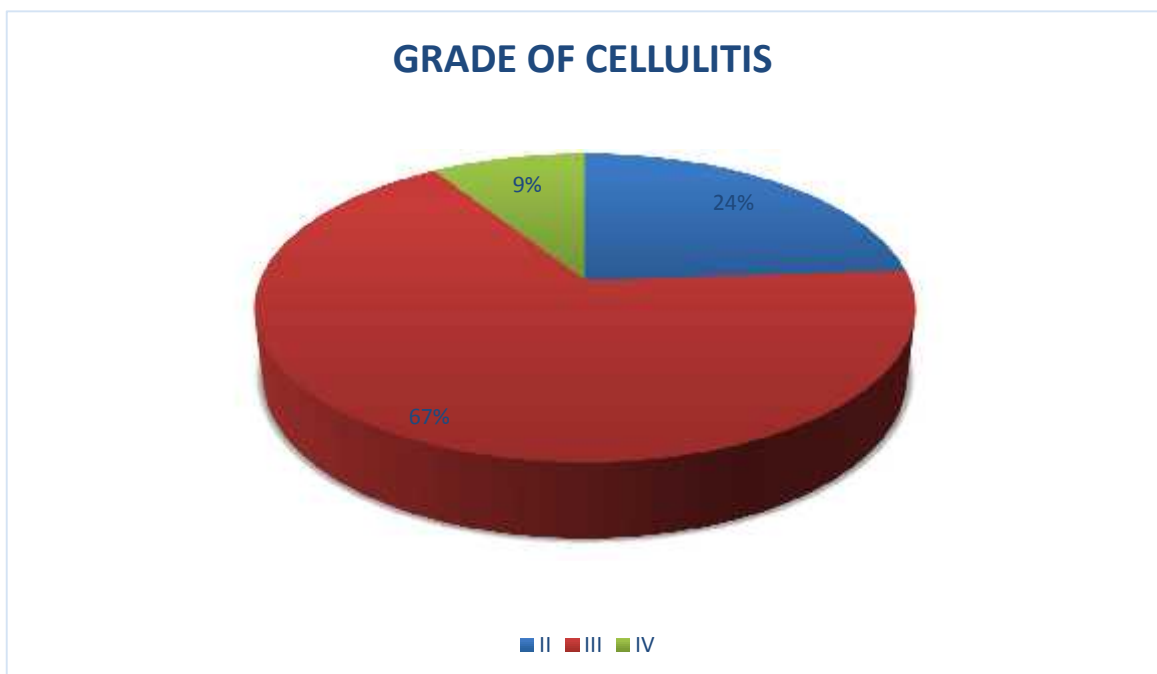
Out of 100 patients 85 patients are male and only 15 patients are females indicating males are more commonly affected.

### 3. GRADE OF CELLULITIS

Grading of cellulitis taken in this study has been done as per CREST criteria. The study is being conducted in the patients who need hospital admission for cellulitis, it covers principally the patients belonging to grades II, III and IV

**TABLE: 3 GRADE OF CELLULITIS**

Sl.No.	Grade	No. of cases
1.	II	24
2.	III	67
3.	IV	9



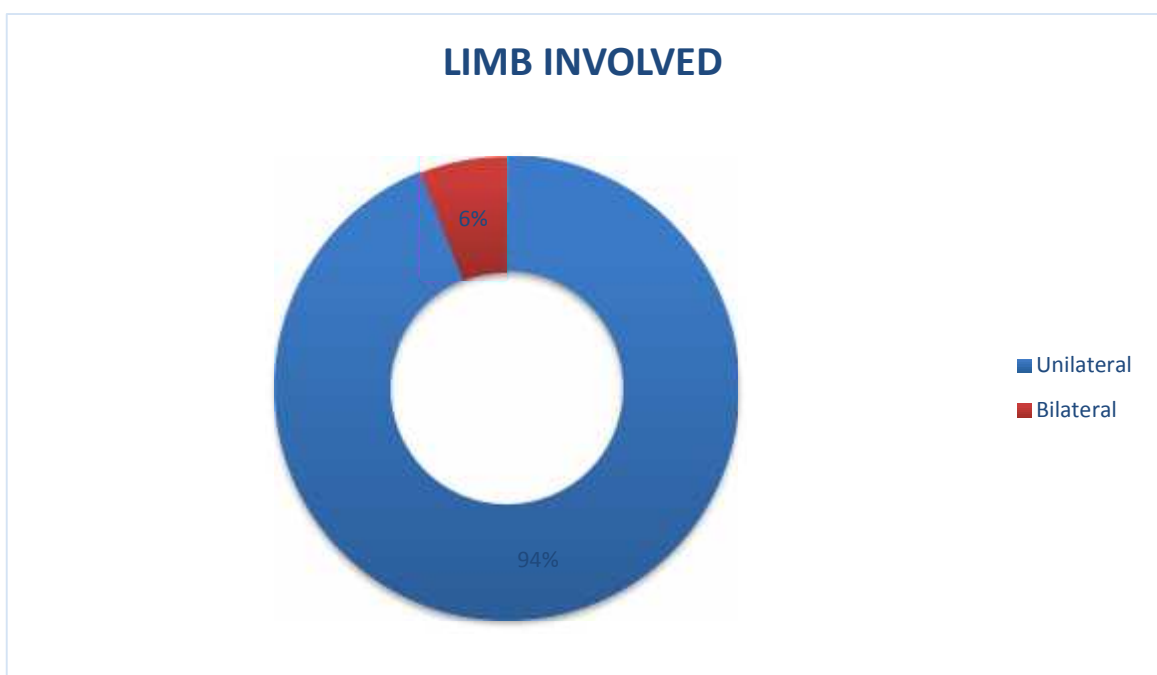
Out of the 100 patients studied maximum number of individuals i.e., 67 individuals belong to the grade III cellulitis, whereas 24 patients and 9 patients belongs to grade II and IV respectively.

#### 4. LIMB INVOLVED

In our study, we have observed that 94 patients had unilateral lower limb involvement and 6 patients had bilateral lower limb involvement, patients with edema going for cellulitis like patients with CKD and cardiac failure, patients with history of barefoot walking with web space infections were candidates who presented with involvement of both lower limbs.

**TABLE: 4 LIMB INVOLVED**

Unilateral	94
Bilateral	6

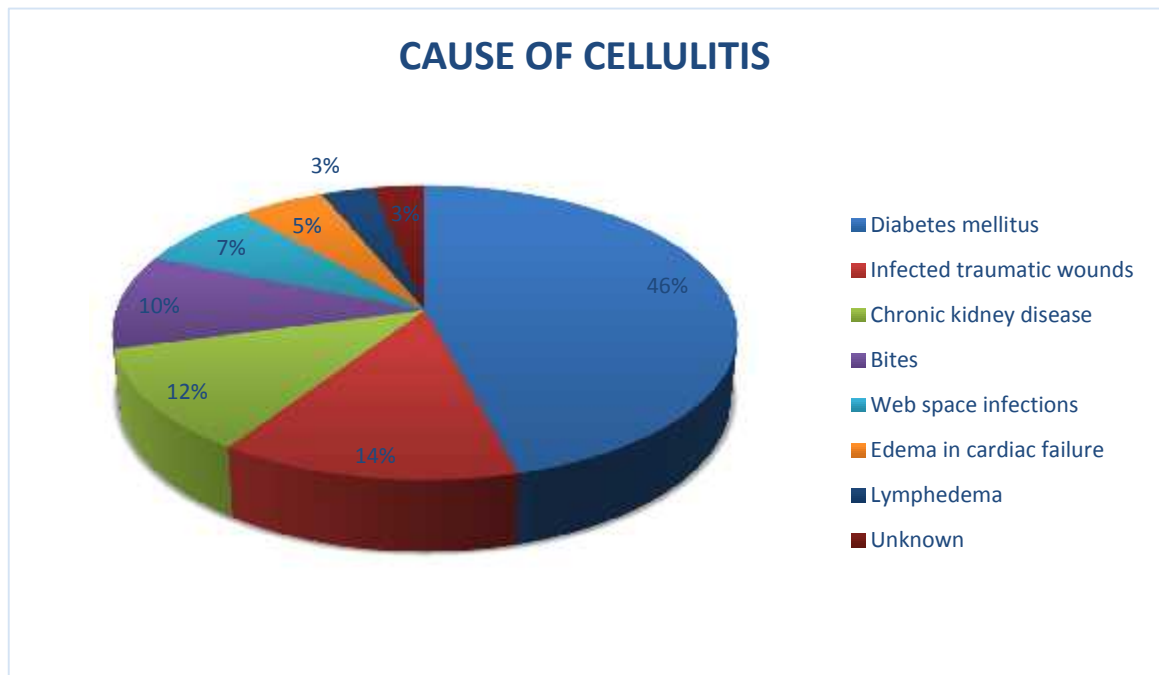


## 5. THE CAUSE OF CELLULITIS:

Various causes were studied for being responsible for the cellulitis in the study group such as web space infections, diabetes mellitus, bites, infected traumatic ulcers, infected venous ulcers, cellulitis imposing on the lymphedematous limb and in the edematous limb of the renal failure and cardiac failure patients, and in few patients exact cause of the cellulitis could not be made. The result can be tabled as follows

**TABLE 5: CAUSE OF CELLULITIS**

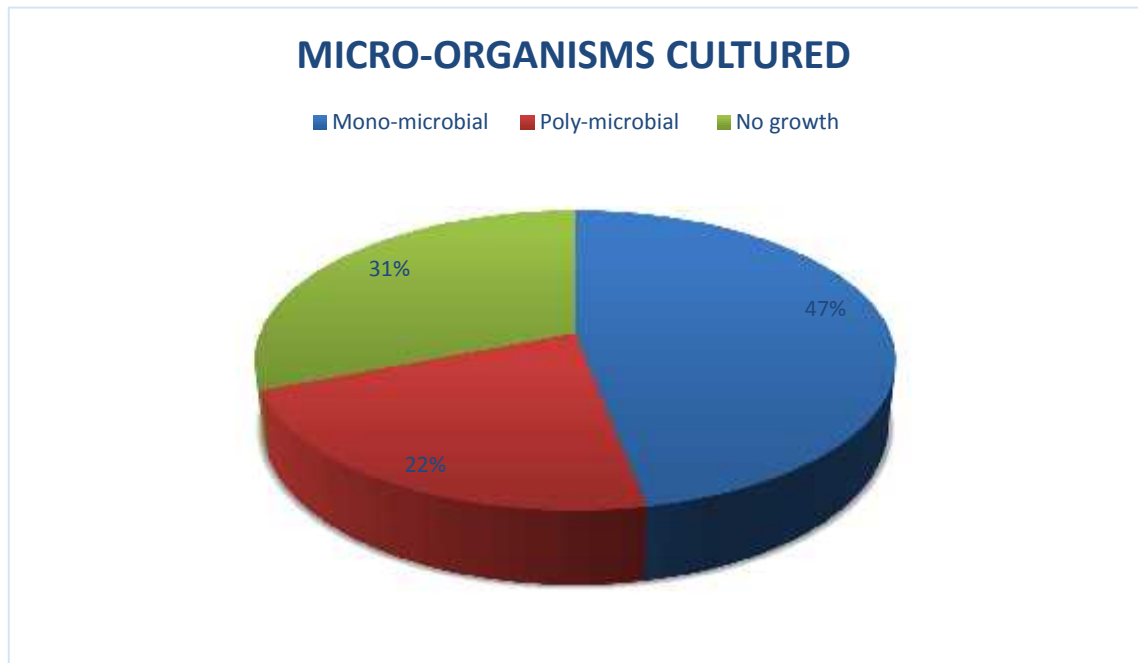
<b>Cause</b>	<b>No. of Patients</b>
Diabetes mellitus	44
Infected traumatic wounds	13
Chronic kidney disease	11
Bites	10
Web space infections	7
Edema in cardiac failure	5
Lymphedema	3
Unknown	3



The chart shows that diabetes mellitus is responsible for most cases of cellulitis in the study group, followed by the traumatic infected ulcers and post bite cellulitis. It is to be noted that cellulitis superimposing on the lower limb edema occurring in chronic kidney disease, lymphedema and cardiac failure constitutes a considerable proportion as the etiology for the cellulitis in our study group. Also in about 3% individuals the exact cause responsible for the cellulitis is unknown.

## 6. MICRO-ORGANISMS CULTURED

Of the 100 patients studied in 47 patients the infection is mono-microbial, in 22 patients the infection is poly-microbial and in about 31 patients no growth has been cultured.

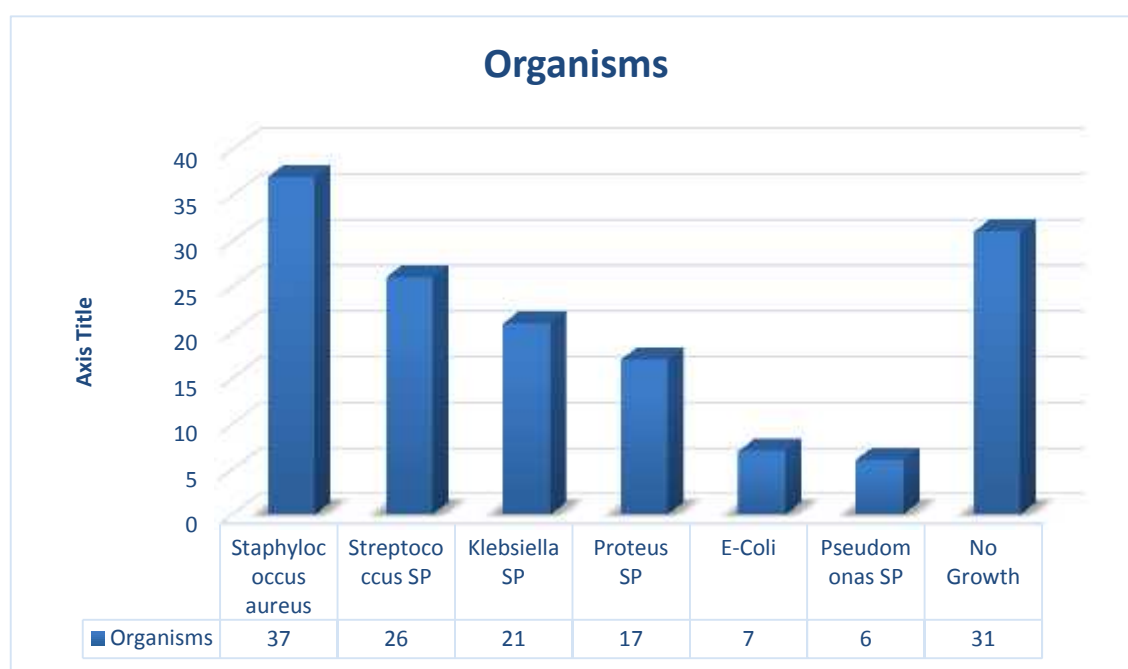


The individual organisms cultured is tabled below

**TABLE: 6 MICRO-ORGANISMS CULTURED**

Sl.No.	Organisms	No. of Patients
1.	Staphylococcus aureus	37
2.	Streptococcus SP	26
3.	Klebsiella SP	21
4.	Proteus SP	17
5.	E-Coli	7
6.	Pseudomonas SP	6
7.	No Growth	31

There results are charted below





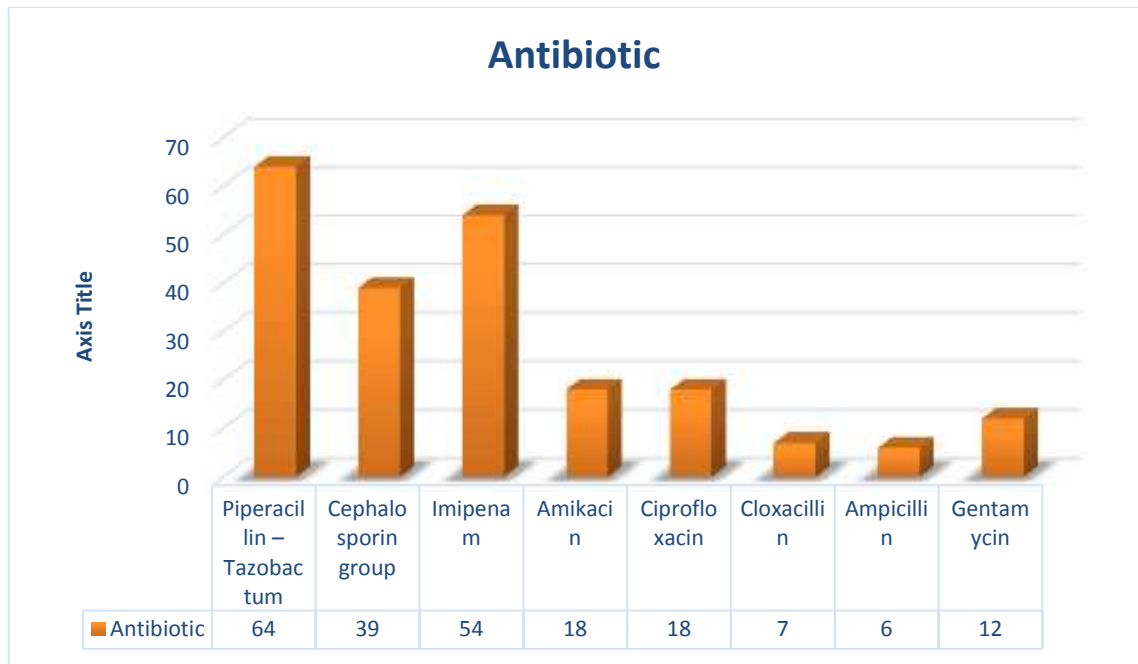
From the table and chart above we can see it is Staphylococcus and streptococcus SP were the predominant organisms responsible for the cellulitis in the study group other organisms responsible include klebsiella SP, proteusSP, pseudomonas SP, Ecoil.

## 7. SENSITIVE DRUGS

Among the 100 patients in our study 69 patients were found culture positive. The sensitivity pattern for the organisms cultured was studied and the results is

**TABLE: 7 SENSITIVE DRUGS**

Sl.No.	Antibiotic	No. of Patients
1.	Piperacillin – Tazobactam	64
2.	Cephalosporin group	39
3.	Imipenam	54
4.	Amikacin	18
5.	Ciprofloxacin	18
6.	Cloxacillin	7
7.	Ampicillin	6
8.	Gentamycin	12



We can see that piperacillin – Tazobactam and imipenem are the two group of antibiotics which tend to have the maximum sensitivity for the common organisms causing cellulitis. The result is plotted on the chart as follows.

## **8. CIRCULATORY CHANGES OBSERVED**

With the Doppler study being conducted in all the patients in our study group, the result obtained is table as follows.

**TABLE: 8 CIRCULATORY CHANGES OBSERVED**

<b>Sl.No.</b>	<b>Changes observed</b>	<b>No. of Patients</b>
1.	No flow in calf vessels	1
2.	Monophasic flow in peroneal artery	4
3.	Monophasic flow in posterior tibial artery	6
4.	Venous insufficiency	4
5.	Deep vein thrombosis	0

## 9. BONE INVOLVEMENT

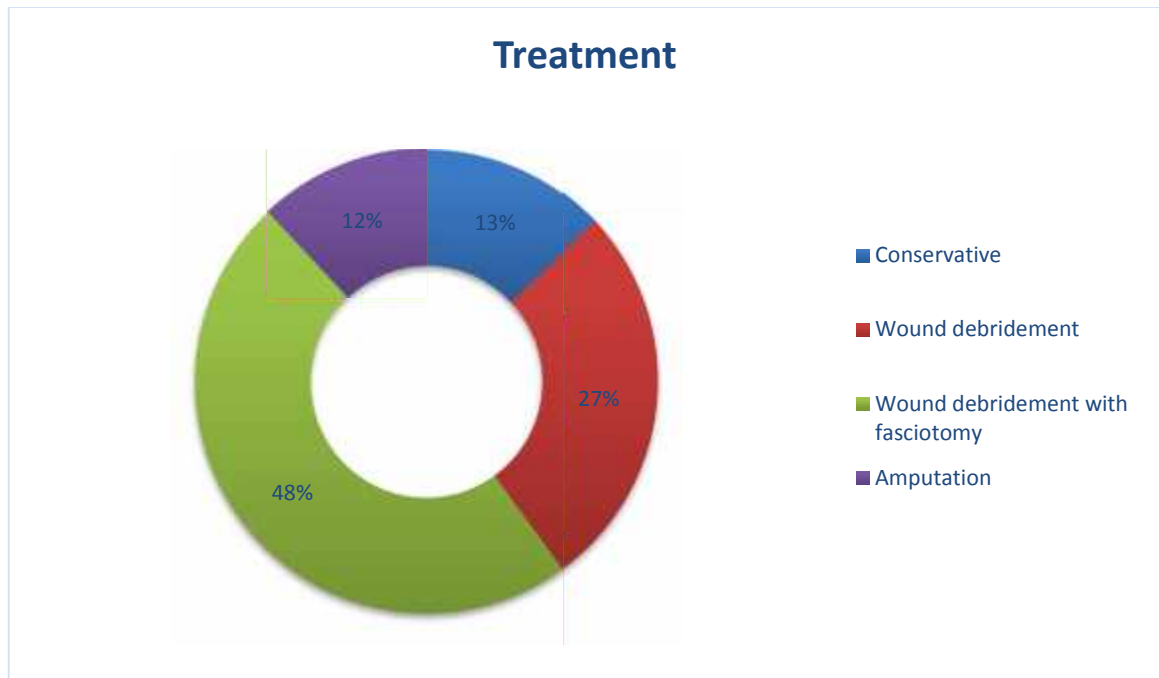
In cases of diabetes mellitus, bite injuries especially in cases of snake bite at site of bite the toes or the metatarsals under neath showed lytic changes, or destruction due to the gangrenous changes otherwise no other bony changes were noticed in the patients.

## 10.TREATMENT

Treatment of the individuals varied according to the severity of the disease, some patients were managed conservatively with parenteral antibiotics, the anti-inflammatory agents and limb elevation so as to reduce the associated edema, while majority of the others required surgical wound debridement with or without decompression of the fascial compartment by a fasciotomy. Very few patients needed amputation of the limb.

**TABLE: 9 TREATMENT**

Sl.No.	Management	No. of cases
1.	Conservative	13
2.	Wound debridement	27
3.	Wound debridement with fasciotomy	48
4.	Amputation	12



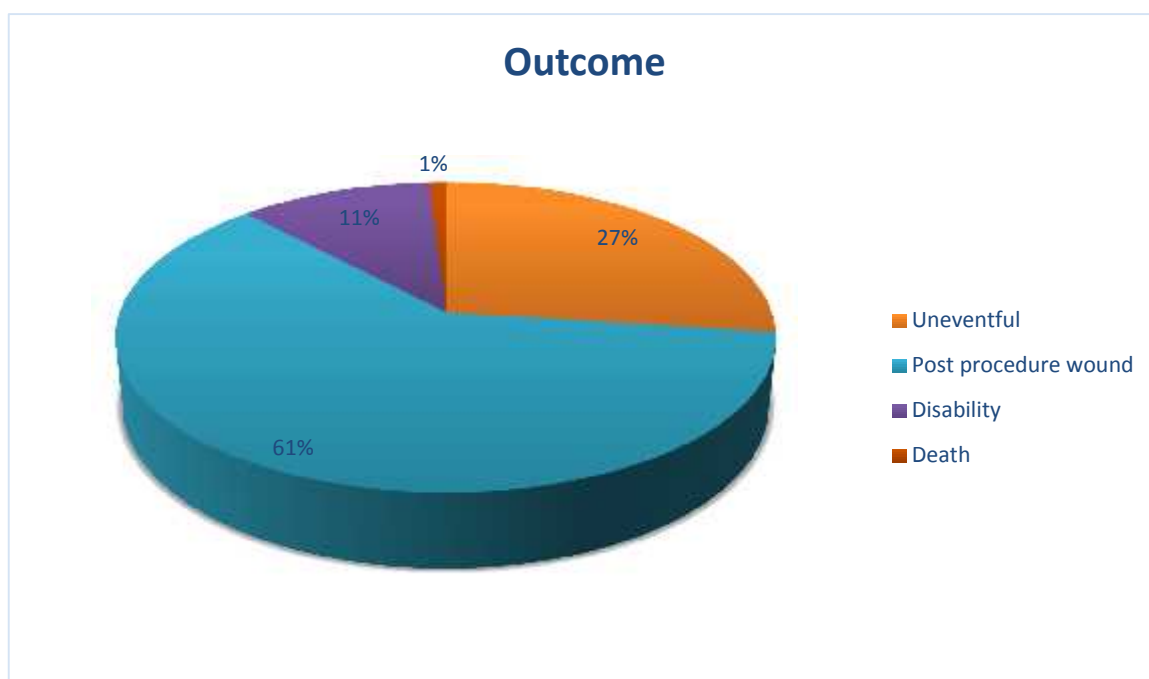
It is observed that around 75 patients in the study group required surgical debridement, 48 of them required decompression of the muscular compartment by means of a fasciotomy. Around 12% of individuals in the study group required amputation.

## 11.OUTCOME

The outcome of the treatment done has been studied, whether it is universal, or whether patient remained with a wound that needs, further managements or patient had some residual deformity or the patient had expired because of the comorbidities complicating the disease.

**TABLE: 11 OUTCOME**

Sl.No.	Outcome	No. of cases
1.	Uneventful	27
2.	Post procedure wound	61
3.	Disability	11
4.	Death	1



It can be seen from the table that almost all the patients managed conservatively, had an uneventful recovery and among those needed surgical intervention, 61 patients had the residual wound that needed further attention, 11 patients remained with disability computation being done and around 1 patient died because of the disease.

This pie chart shows the graphical representation of the observations above. This indicates about the load of patients with residual wound who need further care for the same.



## 12.MANAGEMENT OF THE WOUND

61 patients in the study group with resultant wound were managed ultimately with either a split thickness or allowed to heal by secondary intention.

**TABLE:11 MANAGEMENT OF THE WOUND**

Sl. No.	Management of the wound	No of cases	Percentage
1.	Split skin grafting	17	28
2.	Healing by secondary intention	44	72



It can be observed with this graphical representation that around 28% of the resultant wounds were managed with split skin grafting and remaining 72% of the wounds healed by secondary intention.

## DISCUSSION

Our study, a prospective case series study, included 100 patients who got admitted for lower limb cellulitis and its complications, under all surgical units of Tirunelveli Medical College Hospital, during the span of one year and eight months as the study group. The results observed from the study are discussed here,

Regarding the age distribution, it is evident that as the age increases, the incidence of cellulitis increases, and it has also been studied that the severity of the disease increases with the age, both the inferences correlate, with the literature. This is being explained by the comparatively poorer immune response and the associated comorbidities in the elderly population.

There were 85% males and 15% females, among the 100 patients and this slightly increased male preponderance is supported by the literature.

Out of the 100 patients studied maximum number of individuals, i.e 67 individuals belong to the grade III cellulitis, whereas 24 patients and 9 patients belong to grade II and IV respectively. One consideration to be offered here is, the study is being conducted in the inpatients of the surgical wards, and most early forms of cellulitis are managed on the outpatient basis, our study tend to project the increased incidence of severe forms of cellulitis.

In our study, we have observed than 94% of the patients had unilateral lower limb involvement and 14% of the patients had bilateral lower limb involvement, but according to the literature the incidence of bilateral lower

limb involvement is extremely rare. In our study, cases in whom both lower limb are involved include the patients with edema going for cellulitis like patients with chronic kidney disease and cardiac failure, patients with history of barefoot walking with web space infections, and few patients with unknown etiology.

In our study, incidence wise diabetes mellitus is responsible for most cases of cellulitis in the study group, followed by the traumatic ulcers which have been infected and post bite cellulitis. It is to be noted that cellulitis superimposing on the lower limb edema occurring in chronic kidney disease, lymphedema and cardiac failure constitutes a considerable proportion as the etiology for the cellulitis in our study group. In about 3 individuals the exact cause of cellulitis is unknown.

Of the 100 Patients studied in 47 patients the infection is mono microbial and in 22 patients the infection is poly microbial and in about 31 patients no growth has been cultured. Staphylococcus SP and streptococcus SP were the predominant organisms responsible for the cellulitis in the study group, which correlates with literature. Other organisms observed in the study group include Klebsiella SP, Proteus SP, pseudomonas SP and E coli.

The sensitivity pattern studied for the organisms cultured showed piperacillin tazobactam and imipenam were the two groups of antibiotics which tend to have the maximum sensitivity for the common organisms causing the cellulitis. Cephalosporin group of antibiotics, amikacin,

ciprofloxacin and gentamycin are found to be effective in good proportion of individuals.

All the 100 patients in the study group were done Doppler evaluation of the arterial and venous system to study the circulatory change, associated with the cellulitis of the lower limb. We noticed 4% of the patients showed monophasic flow in peroneal artery and 6% had monophasic flow in posterior tibial artery and venous insufficiency has been noticed in 4% of individuals. 1% of the patients had no flow in the calf vessels and no patients was seen to have deep venous thrombosis.

Bony changes were noticed in the 12% patients, with cellulitis of the concerned limb in phalanges or the metatarsals in cases of diabetes mellitus.

Regarding the treatment, we have noticed that around 75 patients in the study group required surgical debridement, 48 of them required decompression of the some muscular compartment by means of a fasciotomy. 13% of patients with less severe form of cellulitis were managed conservatively with parenteral antibiotics, the anti-inflammatory agents and limb elevation so as to reduce the associated edema 12% of individuals in the study group required amputation, because of the loss of almost all viable soft tissues and the possibility of sepsis syndrome because of the badly infected limb.

Regarding the outcome of the management, almost all the patients managed conservatively had uneventful recovery, around 61% of the

patients had the residual wound that needed further attention, 11% of the patients remained with disability (amputation being done), and around 1% of the patient died because of the comorbidities complicating the illness, especially diabetes mellitus.

In 28% of the patients, resultant wounds persisted as the raw area after preparing the same, they were managed with split skin grafting, and remaining 72% of the wounds were allowed to heal by secondary intention.

## CONCLUSION

- ) As the age increases, the incidence of cellulitis increases and the severity of disease as well.
- ) Males have the higher incidence of cellulitis compared to females.
- ) As our study group is principally comprising of surgical inpatients, higher grades of cellulitis are more common.
- ) In this study, diabetes mellitus, has been the most common cause (overall) of the cellulitis in the patients followed by infected traumatic ulcer and post bite cellulitis.
- ) Staphylococcus SP and streptococcus SP are the common organisms responsible for the cellulitis in the study group, which correlates, with the literature.
- ) Piperacillin tazobactam and imipenam are the most sensitive antibiotics in majority of cases, this shows the emerging resistance for the commonly used antibiotics (ampicillin, cloxacillin and cephalosporins)
- ) Circulatory changes in the form of altered arterial flow pattern has been noticed in 11% of the individuals and in around 4% of the individuals venous reflux has been noticed.
- ) No patient in the study has had deep venous thrombosis.
- ) 12% of the patients showed underlying lytic changes in bone.

- ) 13% of patients were managed conservatively. 75% patients in the study group required surgical debridement, 48% in this group required fasciotomy, and 12% of individuals in the study group required amputation.
- ) All the patients managed conservatively had an uneventful recovery and 61% of the patients had the residual wound that needed further attention 11% of the patients remained with disability, and 1% of the study group patient expired because of the comorbidities complicating the illness.
- ) Majority of the resultant wounds healed by secondary intention 72% rest were managed by split skin grafting 28%.

This study on lower limb cellulitis found that diabetes mellitus is the most common cause besides traumatic infected ulcer, post bite cellulitis, chronic kidney disease also contributing. Early diabetes mellitus screening and good glycaemic control prevent the incidence of cellulitis lower limb.

Educating the people regarding proper foot care, foot wear usage can prevent cellulitis occurring due to web space infections, cracks in the sole, trivial trauma in the foot.

Hospital admission for the severe forms of cellulitis, appropriate and emergency surgical intervention as needed, employing culture directed antibiotics, managing the comorbidities can salvage the limbs and lives.

















## BIBLIOGRAPHY

1. [Guideline] Stevens DL, Bisno AL, Chambers HF, Everett ED, Dellinger P, Goldstein EJ, et al. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. *Clin Infect Dis*. Nov 15 2005;41(10):1373-406.
2. Semel JD, Goldin H. Association of athlete's foot with cellulitis of the lower extremities: diagnostic value of bacterial cultures of ipsilateral interdigital space samples. *Clin Infect Dis*. Nov 1996;23(5):1162-4.
3. Baddour LM, Bisno AL. Non-group A beta-hemolytic streptococcal cellulitis. Association with venous and lymphatic compromise. *Am J Med*. Aug 1985;79(2):155-9.
4. Parada JP, Maslow JN. Clinical syndromes associated with adult pneumococcal cellulitis. *Scand J Infect Dis*. 2000;32(2):133-6.
5. Gray's Anatomy 40<sup>th</sup> edition- *anatomy of the lower limb muscular compartments*
6. Clinical Research Efficiency support Team *guidelines on the management of cellulitis in adults* ISBN 1-903982-12-X
7. Miller LS, Cho JS. Immunity against *Staphylococcus aureus* cutaneous infections. *Nat Rev Immunol*. 2011;11:505-18

8. Hsu PY, Yang YH, Hsiao CH, Lee PI, Chiang BL. *J Formos Med Assoc.* Aug 2002;101(8):581-4.
9. Bassetti S, Battegay M. Staphylococcus aureus infections in injection drug users: risk factors and prevention strategies. *Infection.* Jun 2004;32(3):163-9.
10. Sierra JM, Sanchez F, Castro P, et al. Group A streptococcal infections. *Medicine (Baltimore).* May 2006;85(3):139-46.
11. Horowitz Y, Sperber AD, Almog Y. Gram-negative cellulitis complicating cirrhosis. *Mayo Clin Proc.* Feb 2004;79(2):247-50.
12. Sebeny PJ, Riddle MS, Petersen K. Acinetobacter baumannii skin and soft-tissue infection associated with war trauma. *Clin Infect Dis.* Aug 15 2008;47(4):444-9.
13. Waldhausen JH, Holterman MJ, Sawin RS. Surgical implications of necrotizing fasciitis Aug 1996;31(8):1138-41.
14. Lowy FD. Staphylococcus aureus infections. *N Engl J Med.* Aug 20 1998;339(8):520-32.
15. Barrett FF, McGehee RF Jr, Finland M. Methicillin-resistant Staphylococcus aureus at Boston City Hospital. Bacteriologic and epidemiologic observations. *N Engl J Med.* Aug 29 1968;279(9):441-8.

16. Brook I. Microbiology and management of human and animal bite wound infections. *Prim Care*. Mar 2003;30(1):25-39.
17. Dendle C, Looke D. Review article: Animal bites: an update for management with a focus on infections. *Emerg Med Australas*. Dec 2008;20(6):458-67.
18. TE Whitesides and MM Heckman; Acute Compartment Syndrome: Update on Diagnosis and Treatment; J. Am. Acad. Ortho. Surg., Jul 1996; 4: 209 -218.
19. Steven A. Olson and Robert R. Glasgow; Acute Compartment Syndrome in Lower Extremity Musculoskeletal Trauma; J. Am. Acad. Ortho. Surg., November 2005; 13: 436 – 444
20. Matsen FA 3rd. Compartmental syndrome. An unified concept. *Clin Orthop Relat Res*. Nov-Dec 1975;8-14.
21. Ellis Simonsen SM, van Orman ER, Hatch BE, et al. Cellulitis incidence in a defined population. *Epidemiol Infect*. Apr 2006;134(2):293-9. [[Medline](#)].
22. Lamagni TL, Darenberg J, Luca-Harari B, et al. Epidemiology of severe *Streptococcus pyogenes* disease in Europe. *J Clin Microbiol*. Jul 2008;46(7):2359-67. [[Medline](#)].
23. Medscape, emedicine, Pubmed services for the net references.



## PROFORMA

Name :  
Age :  
Sex : Male Female  
  
Occupation :  
  
Limb involved : Unilateral Bilateral  
  
Symptoms :  
Pain Reddening Swelling Ulcers Blisters  
Bleb  
Comorbid illness :  
Hypertension Epileptic Diabetes Mellitus CKD  
  
CAHD COPD  
  
Previous history of cellulitis:  
Smoker Tobacco user Alcoholic  
  
Cause :  
General Examination  
Nourishment : Hydration status :  
Pallor : Icterus :  
CVS: RS: P/A:  
Vital parameters  
Pulse : BP :  
Temperature : Respiratory rate :  
Examination of limbs:  
  
Extent of cellulitis : Blisters/blebs :  
Subcutaneous abscess : Ulceration :  
Discharge : Healing :  
Deeper tissue involvement: Web space infection:  
Breaks/ Cracks : Distal pulsations :

Colour of the limb	:	Sensation	:
Motor activity	:	Others	:

Lab investigations :

Complete Blood Count

Hb%	:	RBC	:
Differential count	:	Platelet	:
ESR	:		

Blood Sugar

Fasting	:	B. Urea	:
Post-prandial	:	S. Creatinine:	

Culture and sensitivity :

( From the wound either by swab / tissue biopsy )

Liver function test :

Coagulation profile :

HIV ELISA :

HbS Ag serology :

Doppler study of the arterial and venous system :

Plain X-ray of the limb :

Treatment :

### **Conservative**

	Wound debridement
<b>Surgical</b>	Wound debridement & Fasciotomy
	Amputation

Outcome :

Uneventful

Wound to be managed

Disability

Death

Management of the wound:

Split skin grafting

Delayed primary suturing

Healing by secondary intention

## **ABBREVIATIONS**

### **Limb Involvement**

UL - Unilateral

BL - Bilateral

### **Causes:**

B - Bites

DM - Diabetes Mellitus

T - Trauma

V - Venous ulcer

IN - Intertrigo / Web Space Infections

RF - Renal failure

LY - Lymphedema

Un - Unknown Cause

### **Organisms**

St - Streptococcus sp.

S - Staphylococcus aureus

K - Klebsiella

Pr - Proteus sp.

Ps - Pseudomonas Aeruginosa

E - E.coli

NG - No growth

### **Sensitive drugs**

P - Piperacillin-tazobactam

I - Imipenam

Ami - Amikacin

G - Gentamycin

Cip - Ciprofloxacin

Amp - Ampicillin

Clox - Cloxacillin

### **In the Doppler study**

MF - Monophasic flow

NF - No flow

PT - Posterior tibial artery

P - Peroneal

VI - Venous insufficiency

### **X ray-limb**

N - Normal study

### **Regarding management, (Rx)**

WD - Wound debridement

F - Fasciotomy

Con - Conservative

BK amp - Below knee amputation

**Regarding the outcome,**

W - Resultant wound

Un - Uneventful

D - Disability

SSG - Split skin grafting

HS - Healing by secondary intention

SL. NO.	NAME	AGE	SE X	IP NO.	LIMB INV	CAUSE	GRD	CUL ORG	SENSITIVE ANTIBIOTIC	DOPPLER STUDY	PLAIN XRAY	RX	OUT COME	WOUNT MGT
1	Cehlla Pandiyan	62	M	62403	UL	DM	II	NG	-	MF in P	N	C	Un	-
2	Piramudayan	75	M	62414	UL	LY	III	K,Pr,S	P, C, I, G, Ami	N	N	WD	W	SG
3	Karthikeyan	75	M	62479	UL	DM	III	Ps	P, C, I Ami	N	N	WD	W	HS
4	Ramasamy	72	M	62827	BL	DM	III	E	P, G, C, I	N	N	WD, F	W	SG
5	Chellaiah	64	M	71442	UL	CF	III	NG	-	N	N	C	Un	-
6	Mohammed Kani	63	M	74563	UL	RF	III	Ps	P, C, Cip, I	N	N	WD	W	-
7	Aminal	69	F	440	UL	B	III	K,Pr,S	P, C, Ami, G, I	N	N	WD, F	W	-
8	Rahaman Beeri	40	F	77543	UL	T	III	E	P, G, I	N	N	WD, F	W	-
9	Seenirasan	75	M	420	UL	DM	III	K	P, Cip, I	MF in PT	N	WD	W	-
10	Natarajan	48	M	77534	UL	UN	III	S, St	P	N	DEST TOE	A	D	-
11	Chandra	47	F	76096	UL	CF	III	K,Pr,S	P, C, I, Cip, G	N	N	WD, F	W	SG
12	Maharajan	50	M	37124	UL	B	II	NG	-	N	N	C	Un	-
13	Velmayil	75	M	72184	UL	DM	III	Ps	P, C, I, G	N	DEST TOE	A	D	-
14	Chithambaram	70	M	4456	UL	T	IV	E	P, C, I, G	N	N	WD, F	W	SG
15	Esakki	58	M	2850	UL	IN	III	K	G, C	N	N	WD, F	W	-
16	Singaravel	65	M	2778	UL	T	II	S,St	P, C, I	N	N	WD	Un	-
17	Kutty Raj	40	M	71184	UL	DM	III	Ps	P, I, Cip	N	N	WD, F	W	-
18	Esakkimuthu	50	M	69650	UL	DM	III	S	P, C	MF in P	N	WD, F	W	-
19	Vethasingamani Daniel	50	M	71180	UL	DM	III	E	Ami, Cip, C, I	N	DEST TOE	A	D	-
20	Subramaniyan	85	M	71259	UL	DM	III	S	P, C	N	N	WD, F	W	-
21	Govindan	50	M	69671	UL	DM	II	NG-	-	N	N	C	Un	-
22	Rajan	22	M	74494	UL	LY	III	S, St	P, C, I	N	N	WD, F	W	-
23	Ganesan	66	M	74525	UL	RF	III	NG	-	N	DEST TOE	A	D	-
24	Govindaraj	47	M	74515	UL	DM	III	K	P, G, I	MF in PT	N	WD	W	-
25	Subbiah	70	M	71252	UL	DM	III	NG	-	N	N	WD, F	Un	-
26	Ramachandran	60	M	75862	UL	DM	III	K,Pr,S	P, C, I, G, Ami	N	N	WD	W	-
27	Arunachalam	62	M	75090	UL	DM	III	S,St	P, C, I	N	N	WD, F	Un	-
28	Kali	78	M	269	UL	DM	III	S	P, I, Cip	N	N	WD, F	W	-
29	Bakeer Mohamed	54	M	277	UL	DM	III	K,Pr,S	P, C, I, G, Ami	N	N	WD, F	W	-
30	Ramanathan	54	M	4887	UL	DM	III	S	P, C, I	N	N	WD, F	W	-
31	Seeniyeduradiyan	45	M	8205	UL	DM	II	NG	-	N	N	WD	Un	-
32	Murugan	64	M	17718	UL	DM	II	NG	-	N	N	C	Un	-
33	Lambert	47	M	19133	UL	DM	III	S	P, C, I	N	N	WD, F	W	-
34	Irulapasamy	42	M	23859	UL	DM	III	E	Amp, I	N	N	WD, F	Un	-

SL. NO.	NAME	AGE	SEX	IP NO.	LIMB INV	CAUSE	GRD	CUL ORG	SENSITIVE ANTIBIOTIC	DOPPLER STUDY	PLAIN XRAY	RX	OUT COME	WOUNT MGT
35	Ramadurai	58	M	23851	UL	DM	II	S	P, I	N	N	WD	W	-
36	JSokkalingam	46	M	31824	UL	Un	III	NG	-	N	N	WD, F	W	SG
37	Patchathi	40	M	33747	UL	DM	III	E	P, C, Ami, Amp, I	MF in PT	DEST TOE	A	D	-
38	Arunachalam	62	M	33634	UL	DM	III	K,Pr,S,St	P, C, I, Cip, Ami	N	N	WD	W	-
39	Kumaradas	67	M	40282	UL	DM	II	NG	-	N	N	C	Un	-
40	Chelladurai	62	M	43442	UL	DM	III	Ps	P, C, I, Cip	MF in P	N	WD, F	Un	-
41	Kalimuthu	50	M	43444	UL	DM	III	K	P, G, C, I	N	N	WD	W	SG
42	Vellapandi	48	M	45127	UL	DM	II	NG	-	N	N	WD	Un	-
43	Patchaikani	75	M	57129	UL	DM	III	K,Pr,S,St	P, C, I, Cip	N	N	WD, F	W	-
44	Ibrahim	55	M	58130	UL	DM	III	S	P	N	N	WD, F	W	SG
45	Alphonse	56	M	58790	UL	DM	II	NG	-	N	N	C	Un	-
46	Lingam	65	M	58788	UL	DM	III	S,St	P, C, I	N	N	WD	W	-
47	Muthiah	58	M	58831	UL	DM	III	NG	-	N	N	WD, F	Un	-
48	Murugan	62	M	58873	UL	DM	III	S	P, C, Ciox, I	N	N	WD	W	-
49	Karuppasamu	40	M	58885	UL	DM	III	Ps	P, Ami, C, Cip, I	N	N	WD	W	-
50	Iyappan	50	M	60819	UL	DM	III	S	P	N	N	WD, F	W	-
51	Mydeen Pitchai	74	M	64041	UL	DM	II	NG	-	N	N	C	Un	-
52	Arumugam	54	F	63690	UL	DM	IV	K, Pr, S, St	P, C, Ami, Cip, I	N	N	WD, F	W	-
53	Fathima	47	F	63971	UL	DM	III	E	P, Amp, I	N	N	WD	W	-
54	Arumugaradivu	48	F	63999	UL	DM	III	S	P, Ciox, I	N	N	WD, F	W	-
55	Muthiah	58	M	66347	UL	CF	IV	NG	-	N	N	WD, F	W	-
56	Eswari	50	F	68914	UL	T	II	S	P	N	N	WD	Un	-
57	Arumugathamal	75	F	70181	UL	LY	III	K, Pr, S, St	P, C, Ami, Cip, I	N	N	WD, F	W	-
58	Ganapathi	55	M	71993	UL	B	III	NG	-	N	N	WD, F	W	-
59	Mariselvi	20	F	71997	UL	V	II	S	P	VI in SFJ	N	WD	W	-
60	Sornam	60	F	77043	UL	IN	III	ST	P	N	N	WD, F	W	-
61	Balasubramanian	60	M	78749	UL	RF	II	NG	-	N	N	C	Un	-
62	Rajakumar	32	M	78739	UL	V	III	K, Pr, S, St	P, C, Cip, I	VI in SFJ	N	WD	W	-
63	Pitchaiah	63	M	80221	UL	B	III	S	P, I	N	DEST TOE	A	D	-
64	Singarvel	65	M	2778	UL	T	III	NG	-	N	DEST TOE	A	D	SG
65	Chidambaram	70	F	4456	BL	IN	III	K, Pr, S, St	P, C, Ami, Cip, I	N	N	WD	W	-
66	Lakshmanan	75	M	7599	UL	RF	II	S	Ciox, I	N	DEST TOE	A	D	-
67	Balakrishnan	58	M	7716	UL	Un	III	NG	-	MF in P	N	WD, F	W	-
68	Sunramaniyan	62	F	9274	UL	B	IV	S	P, I	N	N	WD, F	Un	SG

SL. NO.	NAME	AGE	SEX	IP NO.	LIMB INV	CAUSE	GRD	CUL ORG	SENSITIVE ANTIBIOTIC	DOPPLER STUDY	PLAIN XRAY	RX	OUT COME	WOUNT MGT
69	Indra	46	M	11022	BL	RF	III	NG	-	N	N	WD	W	-
70	Samsudeen	67	F	11907	UL	DM	II	K, Pr, S, St	P, C, Ami, Cip, I	MF IN PT	N	WD, F	W	SG
71	Ramalakshmi	60	F	12755	UL	CF	III	NG	-	N	N	WD	W	-
72	Mookandi	62	M	53878	UL	B	III	S	P, I	N	N	WD, F	Un	SG
73	Shanmugathai	54	M	14173	UL	DM	IV	K, Pr	P, C, Amp, I	MF in PT	N	WD, F	W	-
74	Veerakumari	55	M	14172	UL	RF	III	S	P	N	N	WD, F	W	-
75	Velladurai	55	M	14256	UL	V	III	NG	-	VI in SFJ	N	C	Un	-
76	Velusamy	70	M	17604	UL	T	II	S	P, I	N	N	WD, F	W	-
77	Shunmugasundram	65	M	17592	UL	B	III	K, Pr, St	P, C, Ami, Cip, I	N	DEST TOE	A	D	-
78	Chellappa	65	M	19384	UL	T	III	NG	-		DEST TOE	WD, F	W	SG
79	Pommiammal	56	F	17632	UL	DM	IV	NG	-	NF in CALF VESSELS	N	BK	D	-
80	Murugan	50	M	52	UL	T	II	St	P, Ciox, I	N	N	WD	W	-
81	Chellappa	64	M	6304	BL	RF	III	St	P	N	N	WD, F	W	SG
82	Esakkiderar	80	M	6370	UL	B	II	NG	-	N	N	C	Un	-
83	Velsamy	60	M	9674	UL	CF	III	St	P	N	N	WD, F	W	SG
84	Madasamy	85	M	11221	UL	RF	IV	NG	-	N	N	WD	W	-
85	Mani	82	M	14429	UL	V	III	K, Pr, St	Ami, C, Cip, Amp, I	VI in SFJ	N	WD, F	W	-
86	Santhanam	67	M	23777	UL	IN	III	St	P, I	N	N	WD, F	W	-
87	Chelladurai	53	M	28395	UL	RF	II	NG	-	N	N	C	Un	-
88	Mohammed Hanifa	90	M	28525	UL	T	III	St	P, I	N	N	WD, F	W	-
89	Thambidurai	52	M	30074	UL	T	III	St	P, I	N	N	WD, F	W	-
90	Paramasivan	60	M	31634	BL	IN	III	S	P, I	N	N	WD, F	W	-
91	Chelladurai	60	M	34657	UL	T	II	St	P, C, Ciox, I	N	N	WD	W	SG
92	Murugan	92	M	38352	UL	B	IV	NG	-	N	N	WD, F	Un	-
93	Kannan	35	M	40112	UL	T	III	St	P, I	N	N	WD, F	W	-
94	Thangapandi	66	M	44881	BL	DM	II	K, Pr, St	P, C, Cip, Ami	MF in PT	N	WD	Un	-
95	Gurusamy	61	M	44910	UL	IN	III	S	P, C, Ami, Ciox, I	N	N	WD, F	W	SG
96	Sivasankaran	60	M	50517	UL	RF	IV	NG	-	N	N	WD, F	Un	-
97	Krishnan	65	M	50534	UL	B	III	K, Pr, St	P, I, Cip, Amp, Ami	N	N	WD	W	SG
98	Eswaran	60	M	50593	UL	T	III	NG	-	N	DEST TOE	A	D	-
99	Murugan	46	M	57069	UL	RF	II	NG	-	N	N	C	Un	-
100	Periyasamy	58	M	60532	UL	T	III	St	P, C, Ciox	N	DEST TOE	A	D	-